February 2025

NASDAQ: **IDYA**

IDEAYA Biosciences

Improving Lives Through Transformative Precision Medicines

Safe Harbor Statement

Certain statements in this presentation and the accompanying oral commentary are forward-looking statements. These statements relate to future events or the future financial performance of IDEAYA Biosciences, Inc. (the "Company") and involve known and unknown risks, uncertainties and other factors that may cause the actual results, levels of activity, performance or achievements of the Company or its industry to be materially different from those expressed or implied by any forward-looking statements. In some cases, forward-looking statements can be identified by terminology such as "may," "will," "could," "would," "should," "expect," "plan," "anticipate," "intend," "believe," "estimate," "predict," "potential" or other comparable terminology. All statements other than statements of historical fact could be deemed forward-looking, assumptions, estimates or projections that are subject to change, including expectations regarding the clinical activity profile, potential clinical benefit and potential advantages of the Company's clinical programs; the translation of preliminary clinical trial results into future clinical trial results; the enrollment of clinical trials;; the potentially addressable patient population for the Company's programs; any expectations regarding the Company's target discovery platform or new target validation efforts as creating opportunities for research and development initiatives; any projections of financial information, market opportunities, cash runway or profitability, including the estimated funding of operations into 2028; any statements about historical results that may suggest trends for the Company's business; any statements of the plans, strategies, and objectives of management for development programs or future operations; any statements about the timing of preclinical research, clinical development, regulatory filings, regulatory approvals, manufacturing or release of data; any statements of expectation or belief regarding future events, potential markets dynamics , technology developments, or receipt of cash milestones, option exercise fees or royalties; and any statements of assumptions underlying any of the items mentioned. The Company has based these forward-looking statements on its current expectations, assumptions, estimates and projections. While the Company believes these expectations, assumptions, estimates and projections are reasonable, such forward-looking statements are only predictions and involve known and unknown risks and uncertainties, many of which are beyond the Company's control. Such risks and uncertainties include, among others, the uncertainties inherent in the drug development process, including the Company's programs' early stage of development, the process of designing and conducting preclinical and clinical trials, serious adverse events, undesirable side effects or unexpected characteristics of drug development, the regulatory approval processes, the timing of regulatory filings, the challenges associated with the manufacturing and/or commercialization; timing of product launches, potential pricing and reimbursement; potential revenue, expected breakrthrough, best or first-in-class or blockbuster status, regulatory landscape, competitive landscape, , the Company's ability to successfully establish, protect and defend its intellectual property, and other matters that could affect the sufficiency of existing cash to fund operations. These and other important factors may cause actual results, performance or achievements to differ materially from those expressed or implied by these forward-looking statements. The forward-looking statements in this presentation are made only as of the date hereof. For a further description of the risks and uncertainties that could cause actual results to differ from those expressed in these forward-looking statements, as well as risks relating to the business of the Company in general, see the Company's periodic filings with the Securities and Exchange Commission (the "SEC"), including its Annual Report on Form 10-K for the year ended December 31, 2024 and any current or periodic reports filed with the SEC. Except as required by law, the Company assumes no obligation and does not intend to update these forward-looking statements or to conform these statements to actual results or to changes in the Company's expectations.

Other

This presentation concerns anticipated products that are under clinical investigation and which have not yet been approved for marketing by the FDA or any other country regulatory authority. These anticipated products are currently limited by Federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company's own internal estimates and research. The Company has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of any information obtained from third-party sources. In addition, all of the market data included in this presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, the Company's own internal estimates and research have not been verified by any independent source.

IDEAYA Biosciences Highlights

Leading Precision Medicine Oncology Biotechnology Company Advancing Potential First-in-Class Therapies

Target Milestone Guidance on Broad Pipeline of 6 Clinical & 3 Preclinical (IND-enabling) Programs:

PHASE 2/3

DAROVASERTIB (PKC)

- Daro + Crizo 1L HLA-A2(-) MUM potential registrational Ph2/3 median PFS readout – by YE 2025
- Daro + Crizo Ph2 1L MUM median OS readout – 2025
- Daro Ph2 Neoadjuvant UM clinical data and regulatory update – H1 2025
- Daro Ph3 Neoadjuvant UM registrational trial initiation – H1 2025

PHASE 1/2

IDE397 (MAT2A)

- Phase 1/2 mono expansion ongoing

IDE397 + Trodelvy® (Trop2-ADC)

- Targeting Expansion in MTAP UC – Q1'25
- Clinical data update in MTAP UC – 2025
- Expansion into NSCLC

IDE397 + PRMT5

- Wholly-owned clinical combo with IDE892 (IDEAYA PRMT5) – H2 2025

IDE849 / SHR-4849 (DLL3 ADC)

- Clinical data update and combo initiation with IDE161 – 2025

IDE275 / GSK959 (WERNER)

- Medical conference update – H1 2025

IDE161 (PARG)

- Phase 1 mono expansion ongoing

IDE161 + Merck's anti-PD-1, KEYTRUDA® (pembrolizumab)

- Phase 1 expansion in EC – 2025

IDE161 + Topo1i-ADC

- Enable clinical combo(s) – 2025

IDE705 / GSK101 (POL THETA)

- Phase 2 expansion ($10M Milestone)

PRECLINICAL

NEXT GEN PROGRAMS

- **IDE892 DC** (MTA-cooperative PRMT5) IND submission – Mid-2025
- **IDE034 DC** (B7H3/PTK7 Bi-Specific ADC) IND submission – H2 2025
- **IDE251 DC** (KAT6/7) IND submission – H2 2025

Pharma Collaborations

Pfizer | GILEAD | MERCK | HENGRUI | GSK

~$2B in potential milestones

Financials and Investor Relations

~$1.1B to fund operations at least into 2028 [1, 2]

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(1) Includes aggregate of $1.1 billion of cash, cash equivalents and marketable securities as of December 31, 2024 (Unaudited)

KEYTRUDA® is a registered trademark of Merck Sharp & Dohme LLC, a subsidiary of Merck & Co, Inc, Rahway NJ, USA

IND = Investigational New Drug, UM = Uveal Melanoma, MUM = Metastatic Uveal Melanoma, NSCLC = Non-Small Cell Lung Cancer, EC = Endometrial Cancer, UC = Urothelial Cancer, DC = Development Candidate, Daro = Darovasertib, Crizo = Crizotinib

IDEAYA BIOSCIENCES

IDEAYA Precision Medicine Oncology Platform

Fully-Integrated Target, Biomarker, Drug Discovery and Translational Capabilities

Target & Biomarker Discovery and Validation

Bioinformatics, including AI Algorithms

Dual CRISPR, CRISPR, Chemogenomics

Genetically Engineered Models

- Key emerging novel targets identified, such as Werner Helicase, PARG and Pol Theta Helicase
- DECIPHER™ - Dual CRISPR SL Library in DDR Cell Lines in collaboration with UCSD
- PAGEO™ - Paralogous Gene Evaluation in Ovarian in collaboration with Broad Institute
- Machine Learning and Multi-Omics platform

Drug Discovery and Pharmacological Validation

Structure Based Drug Design

Small Molecule Chemistry

Protein Degrader Capabilities

- Crystal structures for SL discovery programs obtained to enable structure-based design
- INQUIRE™ Chemical Library - proprietary, expert-curated small-molecule library
- HARMONY™ Machine-Learning engine empowers drug discovery platform
- Differentiated clinical / candidate compounds discovered, including IDE397, IDE275 (GSK959), IDE161, and IDE705 (GSK101)

Translational Research and Opportunity Expansion

Genomics – DNA and RNA Analysis

Proteomics – Protein Expression Profiling

Tissue (IHC, IF) and Liquid Biopsies Analysis

- Translational research to define clinical biomarkers and transformative combinations
- Opportunity expansion through broad cell panel screening
- Pharmacodynamic biomarker analysis to confirm target modulation and correlation with clinical activity

AI = Artificial Intelligence, SL = Synthetic Lethality, DDR = DNA Damage Response, IHC = Immunohistochemistry, IF = Immunofluorescence

IDEAYA Precision Oncology Drug Discovery Platform & IND Engine

AI/ML & Structure Based Drug Design to Deliver Potential First-in-Class Development Candidates

Structural Biology & Structure Based Drug Design

Full suite of capabilities in structural biology, biophysics, & computational chemistry

Ligand bound co-crystal structures resolved to enable Structure Based Drug Design for each of the Synthetic Lethality drug-discovery programs

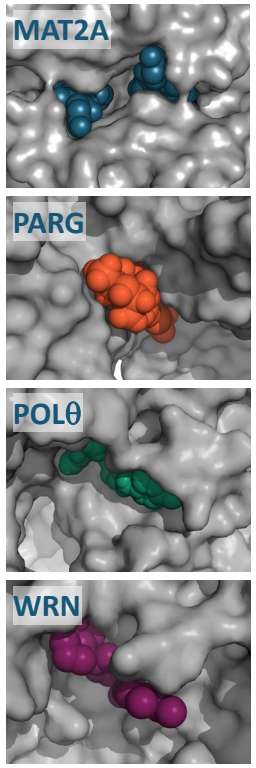
Multiple potential "first-in-world" co-crystal structures resolved, including for Werner Helicase, PARG and Pol Theta Helicase

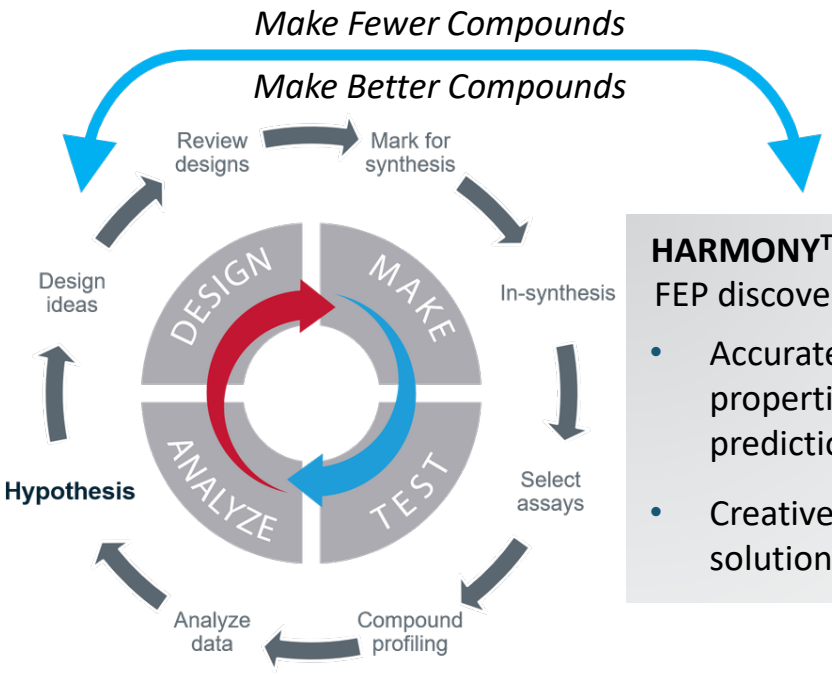
INQUIRE™ Proprietary Chemical Library

Expert-curated HTS library to enhance hit discovery capabilities against novel SL targets classes

Enhances IDEAYA's SL Drug Discovery Platform and competitive differentiation

AI/ML Enabled Computational Drug Discovery[1]





HARMONY™ ML and FEP discovery engine

- Accurate properties prediction
- Creative design solutions

AI/ML to Accelerate Time to IND for Potential First-in-Class DCs

(1) HARMONY™ ML: IDEAYA proprietary AI/Machine Learning platform designed to enable computational drug discovery and lead optimization
HTS = High-Throughput Screening, SL = Synthetic Lethality, DC = Development Candidate, IND = Investigational New Drug, AI/ML = Artificial Intelligence/Machine Learning

IDEAYA's Potential First-in-Class Precision Medicine Oncology Pipeline

	Modality/Indication	Biomarker	Pre-clinical / IND Enabling / Phase 1 / Phase 2 / Potential Registrational	Program Goals / Achievements	Collaborations	Commercial (IDEAYA)
Darovasertib *PKC*	+cMET[1] Combination 1L HLA-A2(-) MUM	GNAQ/11		Ph 2 (AA) / Ph 3 registrational trial[1] – targeting median PFS readout by YE'25	Pfizer (4)	WW Commercial Rights
	(Neo)Adjuvant UM	GNAQ/11		Ph 2 clinical data and reg. update(s) – targeting H1'25 Ph3 Neoadj. UM registrational trial initiation[2] – H1'25		
	cMET[1] Combination MUM	GNAQ/11		Ph 2 OS 1L MUM readout – targeting 2025 HLA-A2(+) Phase 2 clinical trial[3]	Pfizer (4)	
IDE397 *MAT2A*	Monotherapy Solid Tumors	MTAP		Ongoing Phase 2 expansion in MTAP urothelial and lung cancer		WW Commercial Rights
	Combination UC and NSCLC	MTAP		Targeting Phase 1/2 IDE397 + Trodelvy® clinical data update – 2025; expansion into NSCLC	GILEAD (5)	
IDE849 (SHR-4849) *DLL3 ADC*	SCLC, Neuroendocrine Tumors	DLL3		Clinical data update and combo initiation with IDE161 – 2025	HENGRUI (6)	Worldwide Rights Outside of Greater China
IDE275 (GSK959) *Werner Helicase*	Solid Tumors	High-MSI		Ongoing Phase 1 Trial in MSI-High Solid Tumors Medical conference update – 1H'2025	GSK (7)	50% US Profits and 20% costs
IDE161 *PARG*	Monotherapy Solid Tumors	HRD		Ongoing Phase 1/2 expansion in priority tumor type		WW Commercial Rights
	Combination Endometrial Cancer	High-MSI, MSS		Ongoing Phase 1 IDE161 + KEYTRUDA®	MERCK (8)	
IDE705 (GSK101) *Pol Theta Helicase*	+Niraparib Combo Solid Tumors	HR Mutations		Targeting Phase 2 Expansion ($10M Milestone)	GSK (7)	Global Royalties
IDE892 *PRMT5^{MTA}*	Combination Solid Tumors	MTAP		Targeting IND Submission – Mid-Year 2025 Enable wholly-owned combination with IDE397 – H2'2025		WW Commercial Rights
IDE034 *B7H3/PTK7 BsADC*	Solid Tumors	B7H3/PTK7		Targeting IND Submission – H2'2025	BIOCYTOGEN (9)	WW Commercial Rights
IDE251 *KAT6/7*	Solid Tumors	8p11		Targeting IND Submission – H2'2025		WW Commercial Rights
Platform	Solid Tumors	Defined Biomarkers		Multiple Potential First-in-Class Programs Advancing		WW Commercial Rights

(1) Integrated Phase 2/3 enables potential Accelerated Approval (AA, Phase 2) and potential Full Approval (Phase 3) based on FDA Type C Meeting Q1 2023
(2) Phase 3 randomized registrational trial enables potential approval based on FDA Type C Meeting Q3 2024
(3) Targeting enrollment of additional HLA-A2(+) patients in ongoing IDE196-001 Phase 2 clinical trial
(4) Pursuant to Pfizer Clinical Trial Collaboration and Supply Agreements for Darovasertib/Crizotinib Combination; IDEAYA retains all Darovasertib Commercial Rights
(5) Pursuant to Gilead Clinical Study Collaboration and Supply Agreement for IDE397 + Trodelvy®, a Trop-2 directed antibody-drug conjugate (ADC); the Company will sponsor the study and Gilead will provide Trodelvy at no cost. Gilead retains all commercial rights to Trodelvy.
(6) Pursuant to exclusive license agreement with Jiangsu Hengrui Pharmaceuticals Co., Ltd for worldwide rights outside of Greater China
(7) Pursuant to GSK Collaboration, Option and License Agreement: Polθ: Global Royalties; WRN: 50/50 US Profits + ex-US Royalties
(8) Pursuant to Merck Clinical Trial Collaboration and Supply Agreement for IDE161 + Keytruda®, an anti-PD-1 therapy; the Company will sponsor the study and Merck will provide Keytruda at no cost
(9) Pursuant to exclusive worldwide licensing and option agreement with Biocytogen

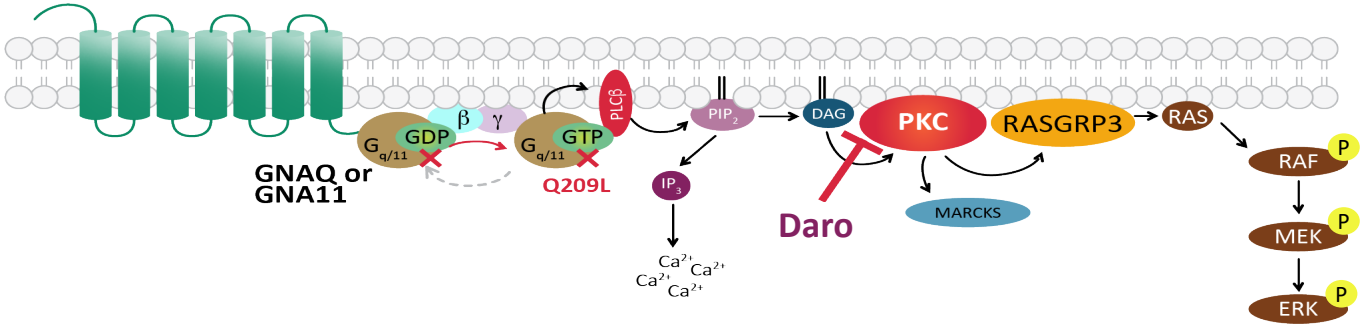
= Target Program Milestones

MAT2A = Methionine Adenosyltransferase 2a, MTAP = Methylthioadenosine Phosphorylase, MTA = Methylthioadenosine, PRMT5 = Protein Arginine Methyltransferase 5, PARG = Poly (ADP-ribose) Glycohydrolase, WRN = Werner Helicase, Polθ = DNA Polymerase Theta, HRD = Homologous Recombination Deficiency, MSI = Microsatellite Instability, PKC = Protein Kinase C, MUM = Metastatic Uveal Melanoma, UM = Uveal Melanoma, Crizo = Crizotinib, UC = Urothelial Cancer ,NSCLC = Non-Small Cell Lung Cancer, WW = Worldwide, HLA-A2(-) = HLA-A2*02:01 Negative; HLA-A2(+) = HLA-A2*02:01 Positive, DC = Development Candidate, TOP1i = Topo-I-Payload, BsADC = Bispecific Antibody Drug Conjugate

Darovasertib: Potential to Broadly Impact Uveal Melanoma (UM)

Potential First-in-Class and Best-in-Class in (Neo)adjuvant UM and Metastatic UM (MUM)

Mutations in GNAQ / GNA11 activate PKC Signaling, a genetic driver of Uveal Melanoma



Darovasertib is an oral, potent and selective PKC inhibitor

GNAQ or GNA11 (~95%) and other upstream mutations activate PKC signaling in UM and MUM patients

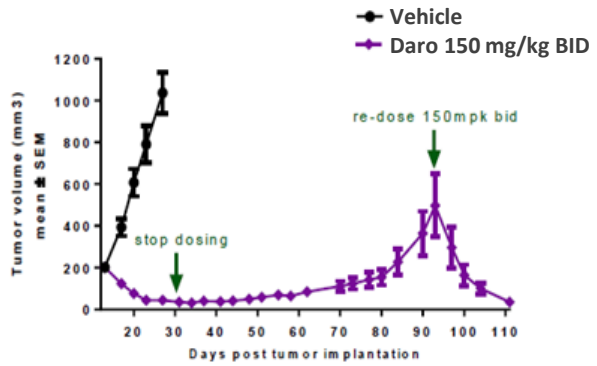
UM is typically treated with radiation and/or enucleation, with no approved systemic therapies for Neoadjuvant UM

MUM occurs in approximately 50% of UM patients and predominantly as liver metastasis in ~90% of MUM patients, with no approved therapies for HLA-A*02:01 negative MUM

Daro Mono Rationale in Primary UM

Single Agent Daro Induces Tumor Regression

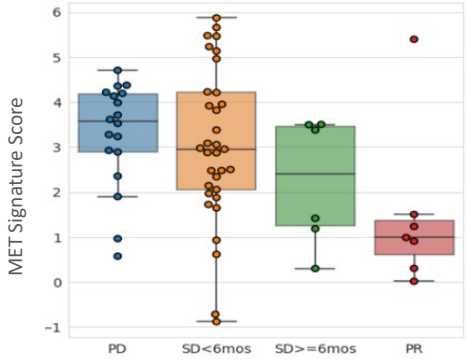
Uveal Melanoma Xenograft (92.1 mutant GNAQ)



Van Raamsdonk, CD, et. al, Nature 2009; Van Raamsdonk CD, et. al, NEJM 2010; Piperno-Neumann S, et. al, J Clin Oncol 2014

Darovasertib + Crizotinib (Daro + Crizo) Combo Rationale for Use in MUM

Daro Phase 1 Monotherapy Efficacy Association with cMET Expression

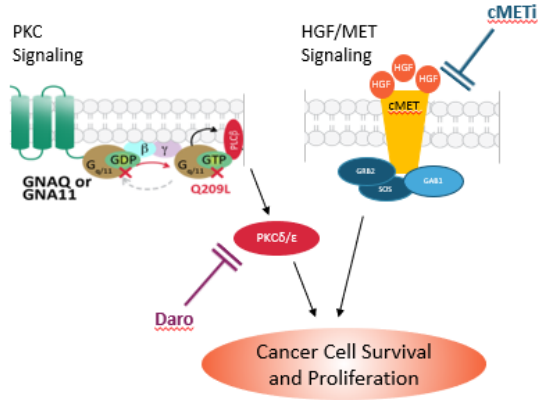


Ph 1 Clinical Outcomes

PD=Progressive Disease, SD=Stable Disease, PR=Partial Response

IDEAYA Data, AACR 2021

Activation of PKC and cMET Pathways with Observed cMET Overexpression in MUM Liver Metastases



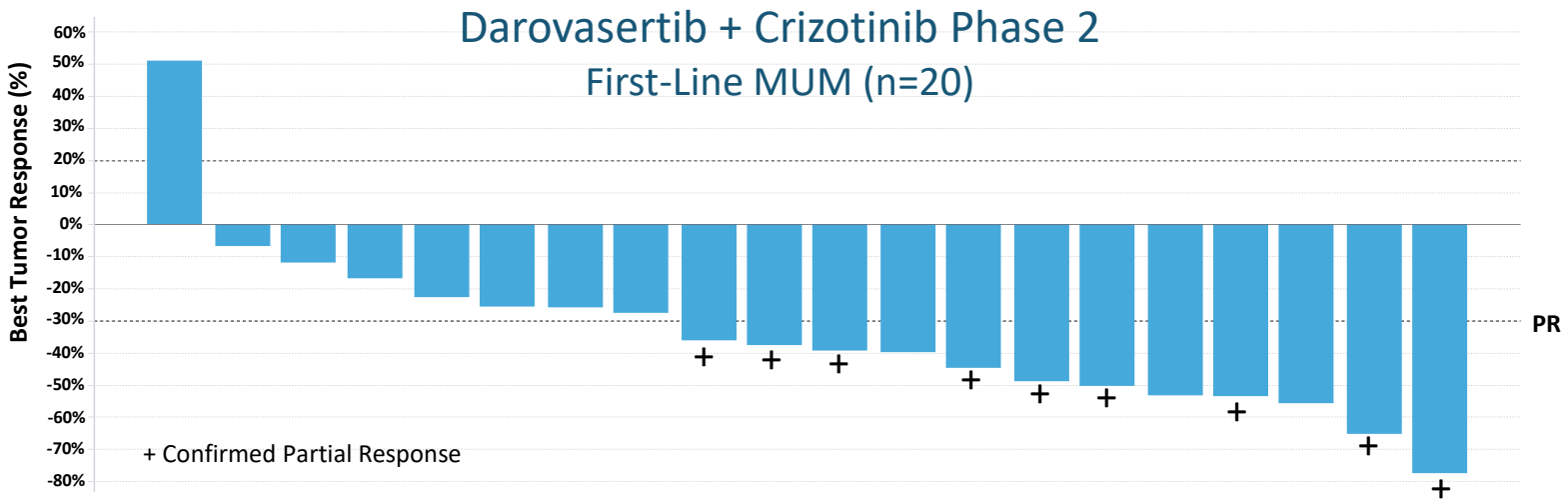
Pfizer [1]

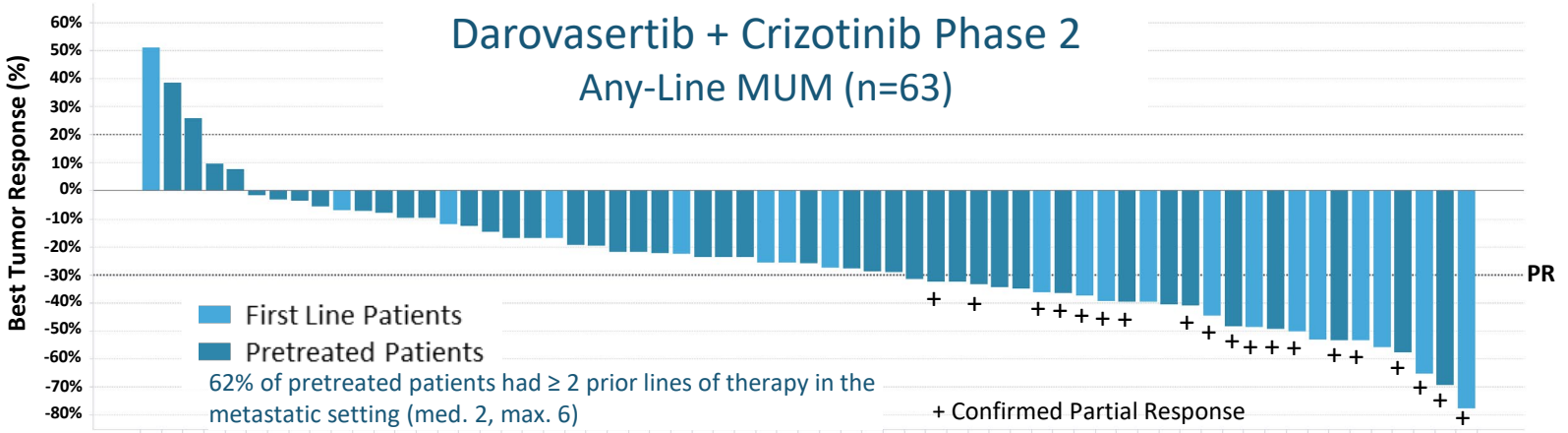
(1) Pfizer Clinical Trial Collaboration and Supply Agreements for Darovasertib + Crizotinib Combination in MUM

IDEAYA owns or controls all commercial rights in darovasertib, including in Primary UM and MUM

Daro + Crizo Phase 2 Efficacy: First-Line MUM and Any-Line MUM

Compelling Overall Response Rate (ORR) by RECIST 1.1 Observed





Confirmed 45% ORR and 90% DCR

Response by RECIST 1.1 First-Line MUM	Evaluable (N=20)
Confirmed ORR (9/20)	45%
Tumor Shrinkage (19/20)	95%
>30% Tumor Shrinkage (12/20)	60%
Best Overall Response	
cPR (9/20)	45%
SD (9/20)	45%
DCR (18/20)	90%

Confirmed 30% ORR and 89% DCR

Response by RECIST 1.1 Any-Line MUM	Evaluable (N=63)
Confirmed ORR (19/63)	30%
Tumor Shrinkage (58/63)	92%
>30% Tumor Shrinkage (27/63)	43%
Best Overall Response	
cPR (19/63)	30%
SD (37/63)	59%
DCR (56/63)	89%

ESMO 2023 Proffered Presentation McKean, M, et al: preliminary analysis of unlocked database as of 8/22/2023 by investigator review, C1D1 cutoff as of 9/22/2022, based on 20 evaluable 1L MUM patients and 63 evaluable Any-Line (includes 1L and 2L+/pre-treated) MUM patients

DCR = Disease Control Rate, cPR = Confirmed Partial Response, uPR = Unconfirmed Partial Response, SD = Stable Disease

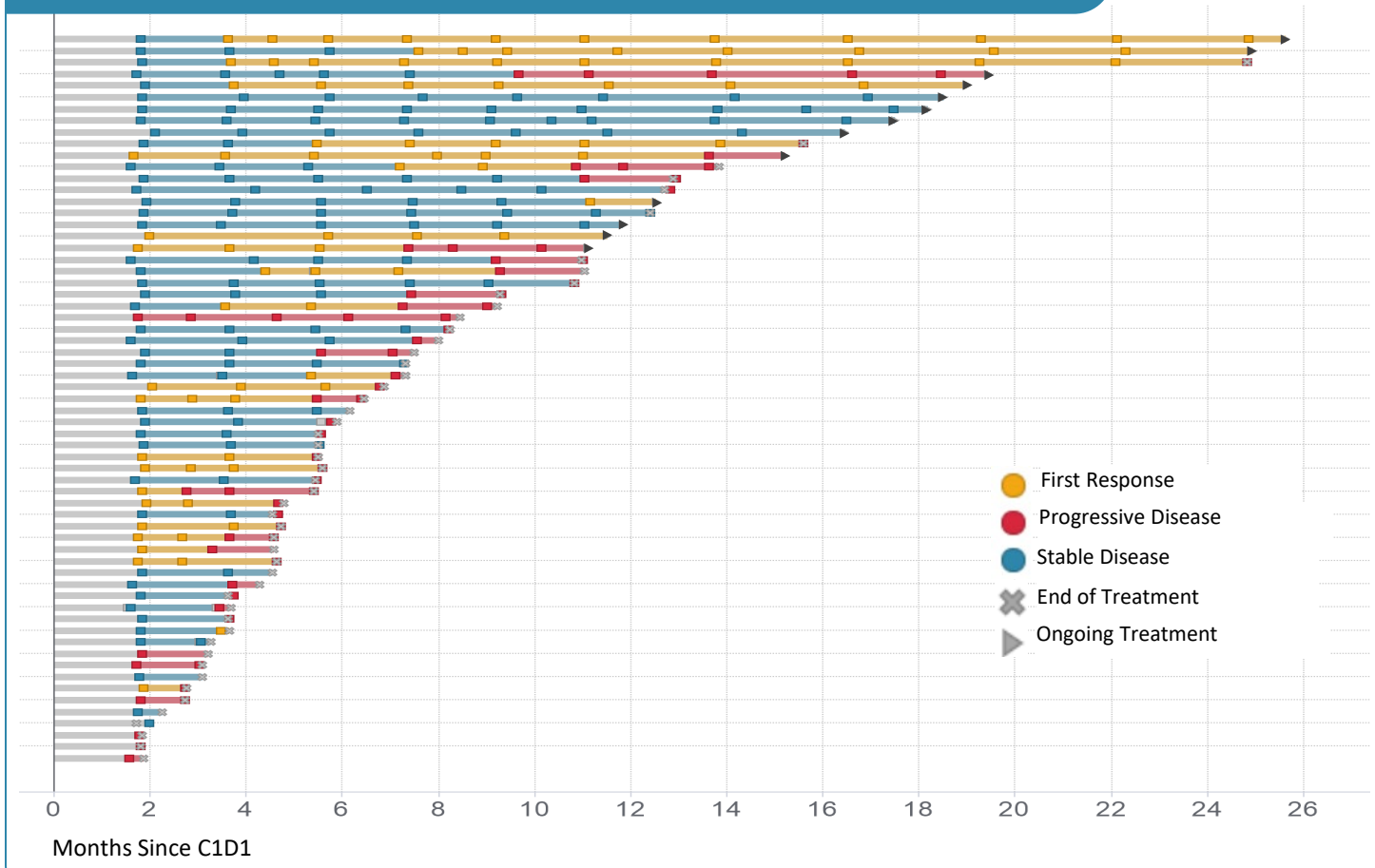
MADRID 2023 ESMO congress

Oral Presentation

Median PFS in First-Line, Any-Line and Hepatic-Only MUM

Observed Compelling Median Progression Free Survival with Encouraging Trend

MUM Swimlane Plot



Darovasertib + Crizotinib Phase 2

Median Progression Free Survival

- First-Line (n=20): 7.1 months
- Any-Line (n=63): 6.8 months
- Hepatic-Only (n=19): 11.0 months

Treatment Duration – Observations

- ~50% of patients treated > 6 months
- ~30% of patients treated > 1 year

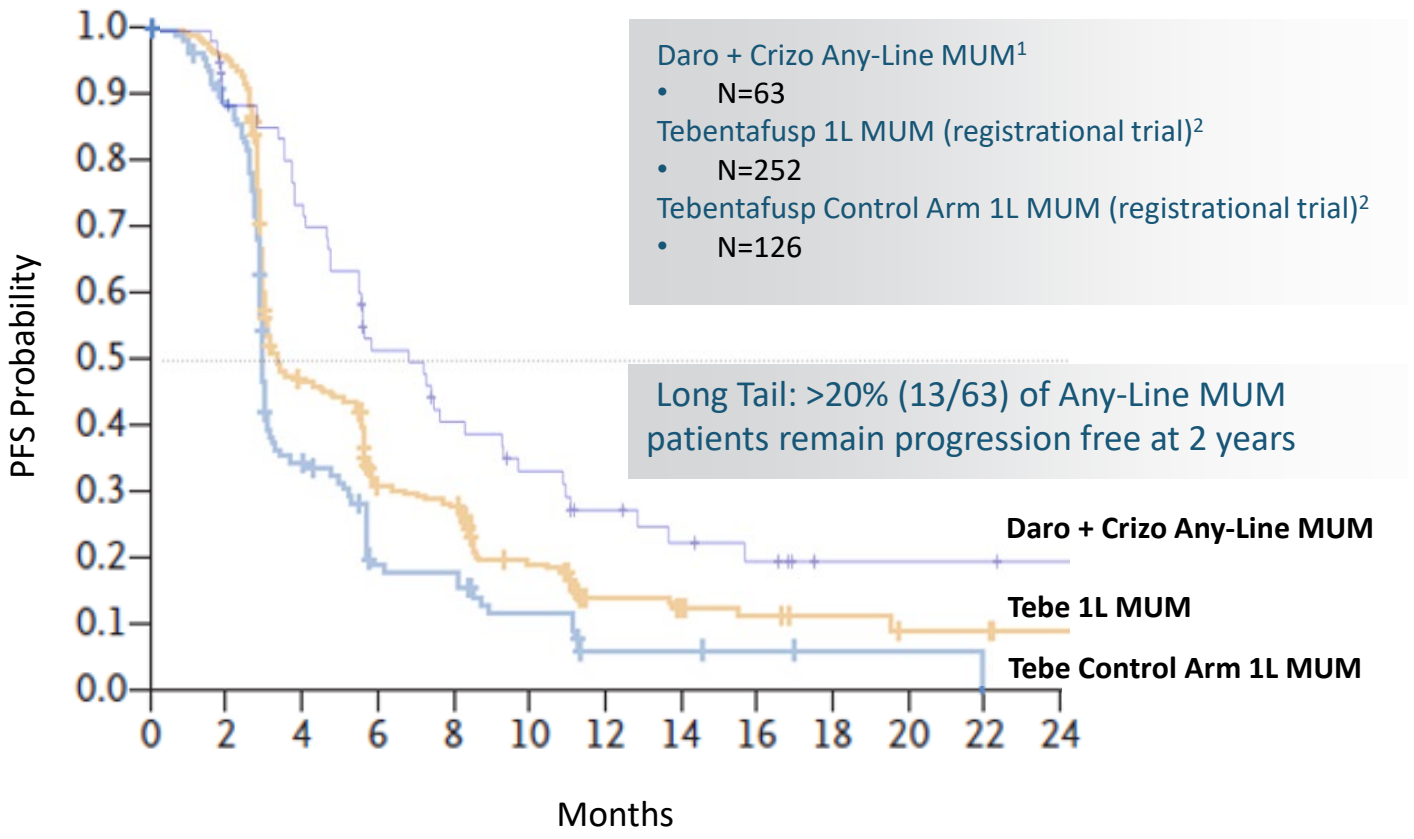
ESMO 2023 Proferred Presentation M McKean et al: Preliminary analysis of unlocked database as of 08/22/2023 by investigator review, C1D1 cutoff as of 9/22/2022, based on 63 evaluable Any-Line MUM patients

IDEAYA BIOSCIENCES

2-Year PFS Kaplan Meier (KM) Curve: Daro + Crizo in Any-Line MUM[1]

Daro + Crizo Combo Observes a Promising 2-Year PFS KM Curve and a "Long Tail" Effect

2-Year PFS Kaplan Meier Curves in MUM



Daro + Crizo Any-Line MUM[1]
- N=63

Tebentafusp 1L MUM (registrational trial)[2]
- N=252

Tebentafusp Control Arm 1L MUM (registrational trial)[2]
- N=126

Long Tail: >20% (13/63) of Any-Line MUM patients remain progression free at 2 years

(1) IDEAYA Data: preliminary analysis of unlocked database as of 08/22/2023 by investigator review, C1D1 cutoff as of 9/22/2022, based on 63 evaluable Any-Line MUM patients. Direct comparisons are not being made and the historical data for tebentafusp is being shared for informational purposes only

(2) N Engl J Med 2021;385:1196-206; Tebentafusp Phase 3 registrational trial, PFS curves

Darovasertib + Crizotinib Combination Clinical Summary in MUM

Highly Differentiated Clinical Efficacy & AE Profile Observed[1, 2]

	Darovasertib + Crizotinib	Cabozantinib Mono / Crizotinib Mono	Selumetinib + DTIC	Ipi + Nivo	Tebentafusp
Target / Mechanism	PKC + cMET	cMET	MEK + Chemotherapy	CTLA4 + PD-1	HLA-A2-0201 Bi-Specific
Study Name(s)	NCT03947385	A091201[3] / NCT05063058[4]	NCT01974752[5]	NCT02626962[6]	IMCgp100-102[7]
Population	1L/2L/3L+ MUM (n=63)	1L+ MUM (n=31) / 1L (n=6) 2L (n=1) MUM	1L+ MUM (n=97)	1L MUM (n=52)	2L+ MUM (n=127)
Patient Selection	NA	NA / MET Overexpression	NA	NA	HLA-A2-positive
Drug Form	Oral Tablets	Oral Capsules	Oral Capsules + chemo	IV infusion	IV Infusion (Weekly)
Tolerability (Grade ≥3 Drug-Related AE)	31%	51.6% / NA	63% (All Cause)	58%	46.5%
% of Patients with Tumor Shrinkage	First-Line = 95% / Any-Line = 92% / Hepatic Only = 100%[8]	23%[9] / NA	35%[9]	27%[9]	44%[9]
Confirmed ORR% (by RECIST 1.1)	**First-Line = 45% / Any-Line = 30% / Hepatic Only = 37%[8]**	**0% / 0%**	**3%**	**11.5% (not confirmed ORR)**	**4.7%**
Median PFS	**First-Line: 7.1 months / Any-Line: 6.8 months / Hepatic-Only: 11.0 months[8]**	**2 months / NA**	**2.8 months**	**3 months**	**2.8 months**

(1) Cross-trial comparisons are not based on head-to-head studies and are presented for informational purposes; no direct comparisons are being made
(2) ESMO 2022: Dimitriou, F, et. al: IPI + Nivo Combo in HLA-A2-0201 MUM reports ~6% ORR (2 PRs out of 33 patients)
(3) Randomized Phase II Trial and Tumor Mutational Spectrum Analysis from Cabozantinib versus Chemotherapy in Metastatic Uveal Melanoma (Alliance A091201); Clin Cancer Res 2020;26:804–11
(4) European Journal of Cancer, Leyraz, et. al, 2022; 146-155
(5) Journal of Clinical Oncology, Carjaval, et. al, 2018; 1232-1239
(6) ASCO 2021, Piulats, J, et. al, Ipi = Ipilimumab, Nivo = Nivolumab, ORR% did not require PR/CR confirmation
(7) Based on Immunocore reported 2L+ study data (to reflect comparative patient population) and by independent review and ORR% was with confirmed PRs
(8) ESMO 2023 Proffered Presentation McKean, M, et al: Preliminary analysis of unlocked database as of 08/22/2023 by investigator review; data cutoff based on treatment Day 1 of Cycle 1 (C1D1) as of 9/22/2022
(9) Estimated from Waterfall plot

IDEAYA BIOSCIENCES

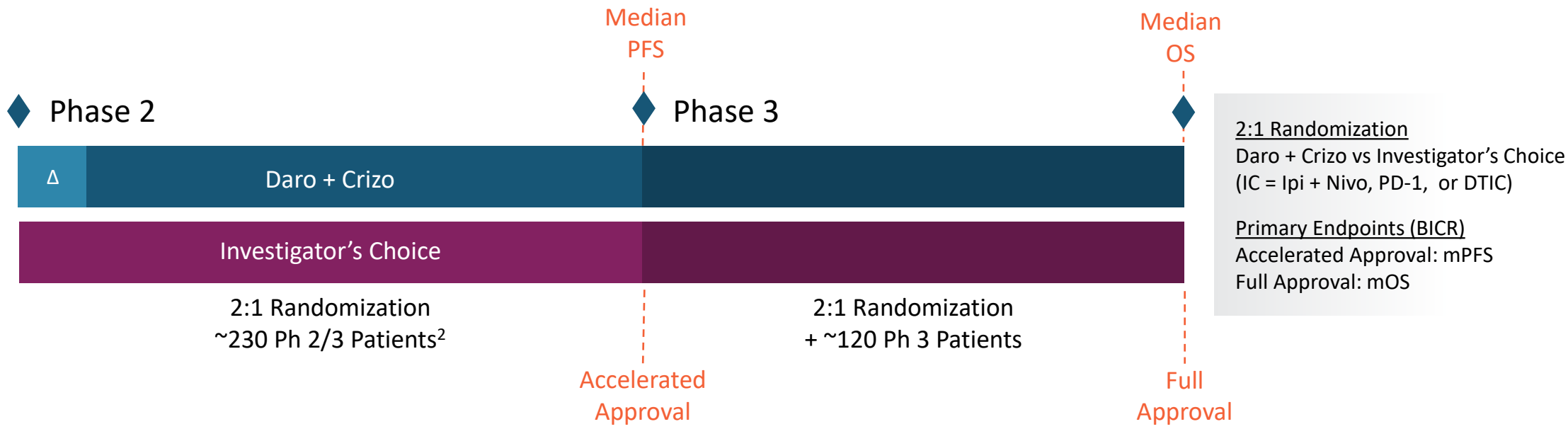
Accelerated Approval Trial Design in First-Line HLA-A2 Negative MUM

FDA Guided Design: Open Label Ph2/3 Evaluation of Daro + Crizo vs. Investigator's Choice[1]

FDA Project FrontRunner: Target First-Line approval strategy to enhance patient benefit in MUM

FDA Accelerated Approval: Address unmet need in MUM, which has limited effective treatment options

Integrated Phase 2/3 Study within Study Enables Potential Accelerated Approval & Full Approval

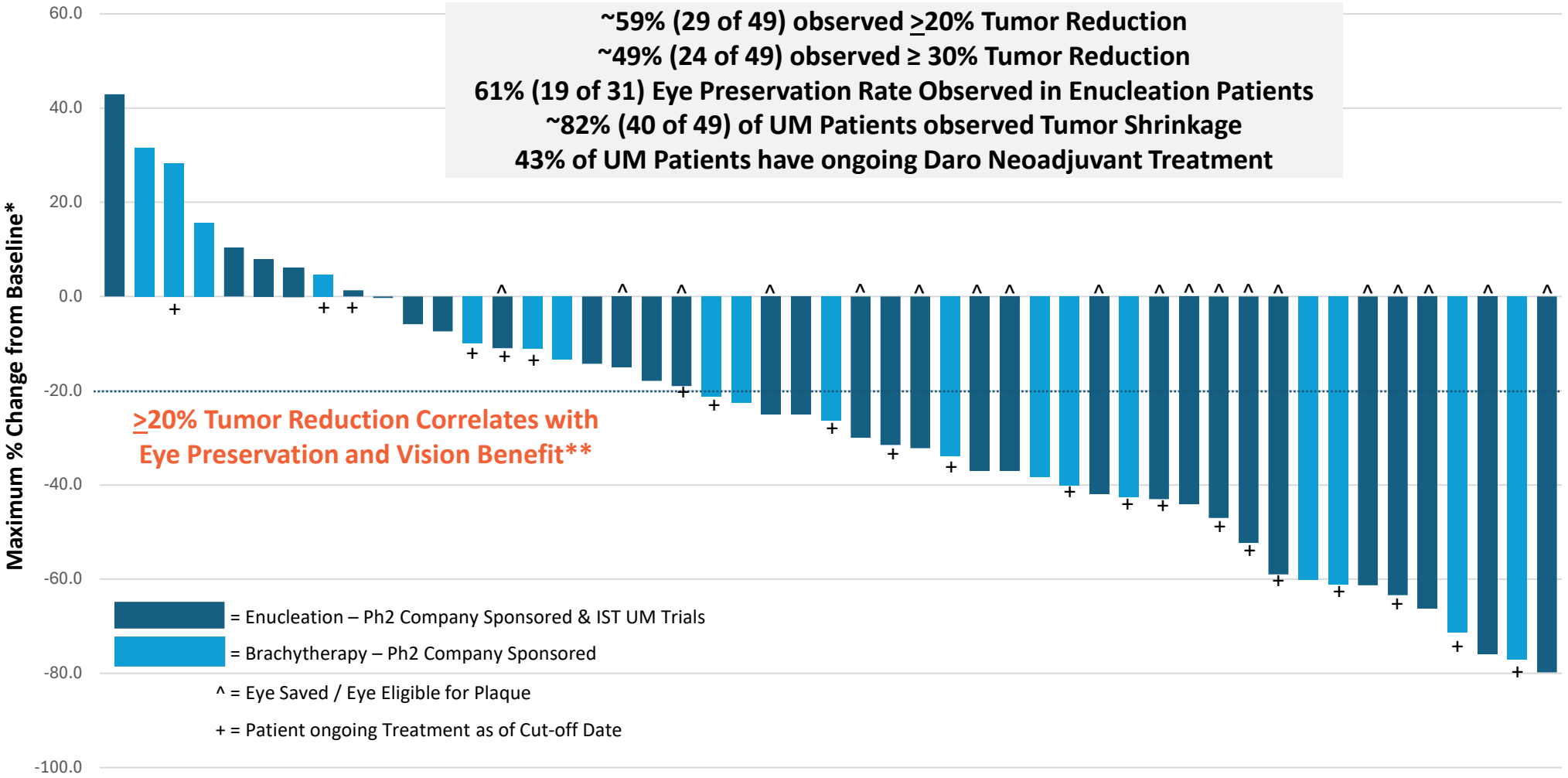


FDA Fast Track and EMA SME Status Designation for Daro + Crizo in MUM

(1) Clinicaltrials.gov: NCT05987332
(2) Phase 2 study contemplates data set of n=200 patients randomized 2:1 with treatment arm at move forward dose in support of potential accelerated approval based on mPFS
Δ Nested study to confirm move forward dose: (i) Daro 300 mg BID + Crizo 200 mg BID or (ii) Daro 200 mg BID + Crizo 200 mg BID
Daro = Darovasertib, Crizo = Crizotinib, MUM = Metastatic Uveal Melanoma, HLA-A2 = HLA-A2*02:01 Serotype, IC = Investigator's Choice, Ipi = Ipilimumab, Nivo = Nivolumab, DTIC = Dacarbazine

Darovasertib Neoadjuvant Therapy: Ph2 Company Sponsored & Ph2 IST UM Trials

61% (19 of 31) Observed Eye Preservation and 49% (24 of 49) with ≥30% Tumor Reduction*

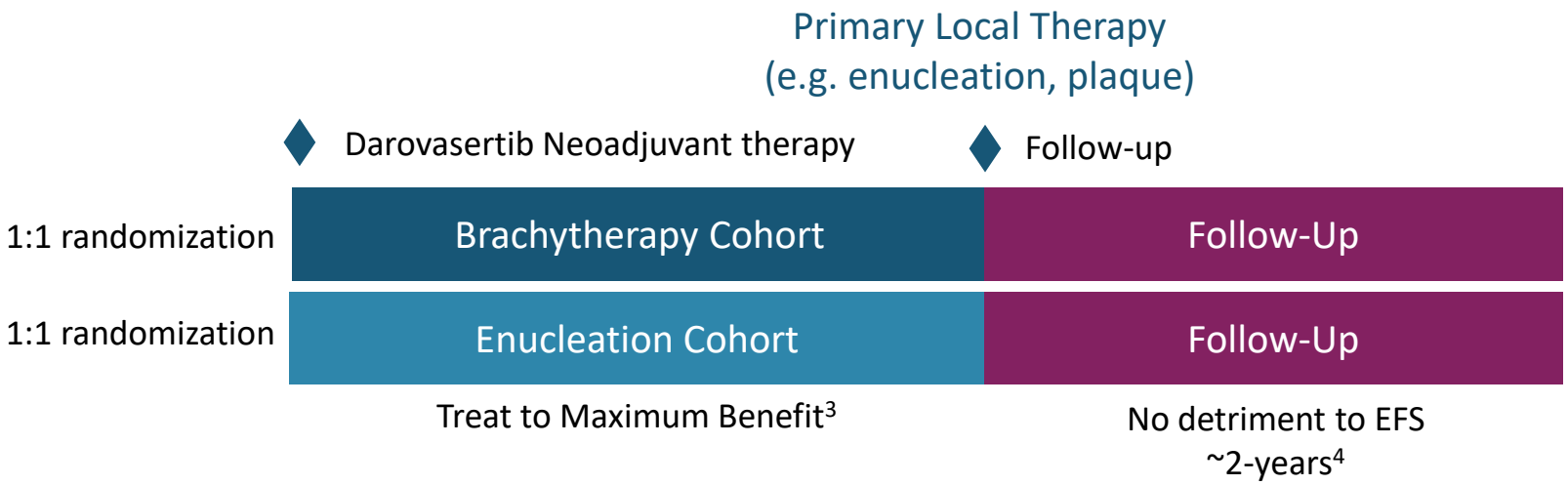


Clinical Efficacy (n=49)	
Evaluable Patients, n	49
% Tumor Reduction, by Product of Diameters measurements, n, (%)	
≥ 20% Reduction	29 (59%)
≥ 30% Reduction	24 (49%)
Eyes Preserved in Enucleation Pts, n, (%)	19/31 (61%)

IDEAYA Data: Enrollment cut-off date of 13May24, and results as of 15Aug2024 (based on preliminary analysis of unlocked database for Ph2 company sponsored patients enrolled up to 13May2024); Ph2 IST as of 14May2024 [ASCO 2024 Oral Presentation]
*Ocular tumor size measured by the product of diameters (longest basal diameter x tumor thickness); **Based on clinical data correlating ocular tumor shrinkage with eye preservation and vision from darovasertib treatment in UM. Clinical data provided in FDA briefing book for FDA Type C meeting
IST = Investigator Sponsored Trial

Preliminary Darovasertib Neoadjuvant UM Phase 3 Trial Design[1]

Paradigm Shifting Opportunity to Save the Eye and Protect Vision



Primary Endpoints[2]

- Cohort 1: Time to Vision Loss
- Cohort 2: Eye Preservation

Secondary Endpoints

- Cohort 1 and 2: No detriment to Event Free Survival (EFS). Initial EFS readout anticipated in ~2-years

FDA discussion ongoing for use of ORR as potential surrogate and composite endpoint for earlier approval scenarios

Three Independent Approaches for Demonstrating Clinical Benefit With Approval Pathway

Enucleation Cohort → Save the Eye Brachytherapy Cohort → Protect Vision Follow-up → No detriment to EFS

(1) Protocol finalization pending FDA Type B meeting
(2) FDA briefing book notes clinical endpoint target to exceed a lower bound of 10% for eye preservation rate with a 95% confidence interval
(3) Treatment to maximum benefit: continued observation of ocular tumor shrinkage
(4) Estimate of initial no detriment EFS readout of UM patients with high risk of metastatic disease

Darovasertib and Uveal Melanoma Patient Journey

High Unmet Need and Multiple First-Line Opportunities in UM and MUM[1]

+95% of UM patients harbor GNAQ/GNA11 mutation

Uveal Melanoma Patient Journey

	Neoadjuvant UM	Adjuvant UM	MUM
HLA-A2-Negative (~70% of UM / MUM)[2]	No FDA Approved Therapies[1]: **Daro** Phase 2/3 Enucleation Define Approval Path; **Daro** Phase 2/3 Radiation Define Approval Path	No FDA Approved Therapies[1]: **Daro** Phase 2	No FDA Approved Therapies[1]: **Daro + Crizo** Registrational Trial Accelerated Approval Full Approval
HLA-A2-Positive (~30% of UM / MUM)[2]			**Daro + Crizo** Target NCCN / Compendia Listing
Target Treatment Duration	≥6 months	≥6 months	mPFS + ~3 months
Target Clinical Endpoints	Eye Preservation, Time to Vision Loss, No detriment to EFS	Relapse Free Survival	ORR, mPFS, mOS
Annual Incidence[3]	~12K	~12K	~4-5k

FDA Orphan Drug Designation in Uveal Melanoma[4]; FDA Fast Track Designation in Metastatic Uveal Melanoma

Phase 2/3 Registrational Trial Ongoing in HLA-A2 negative 1L MUM for both Accelerated and Full Approval

(1) No FDA approved systemic therapies in multiple UM and MUM indications across the patient journey
(2) IDEAYA data: ~70% of MUM patients were HLA-A2-negative based on 149 patients tested for HLA-A2 status as presented at ESMO 2023
(3) Annual incidence for North America, Europe and Australia (as applicable), based on market research analysis
(4) Orphan Drugs benefit from certain tax credits and may be excluded from certain mandatory price negotiation provisions of the 2022 Inflation Reduction Act
UM = Uveal Melanoma, MUM = Metastatic Uveal Melanoma, ORR = Overall Response Rate, mPFS = Median Progression Free Survival, mOS = Median Overall Survival

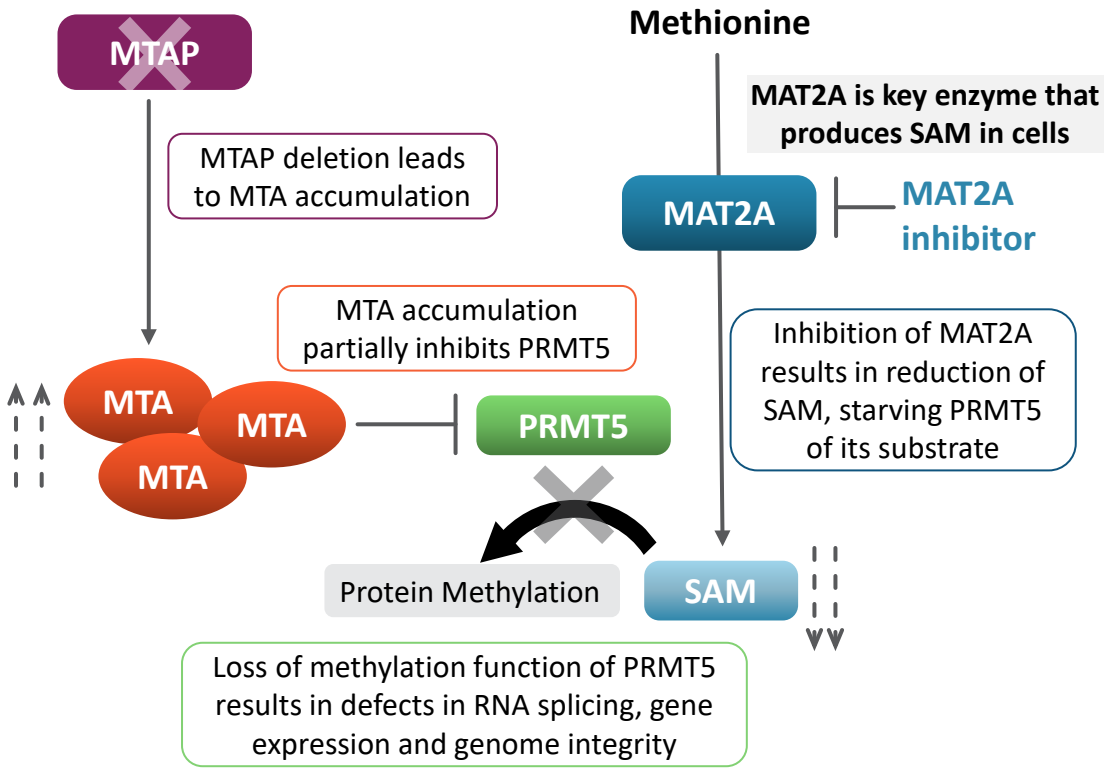
IDEAYA BIOSCIENCES

MAT2A Inhibition is Synthetic Lethal with MTAP-Deletion

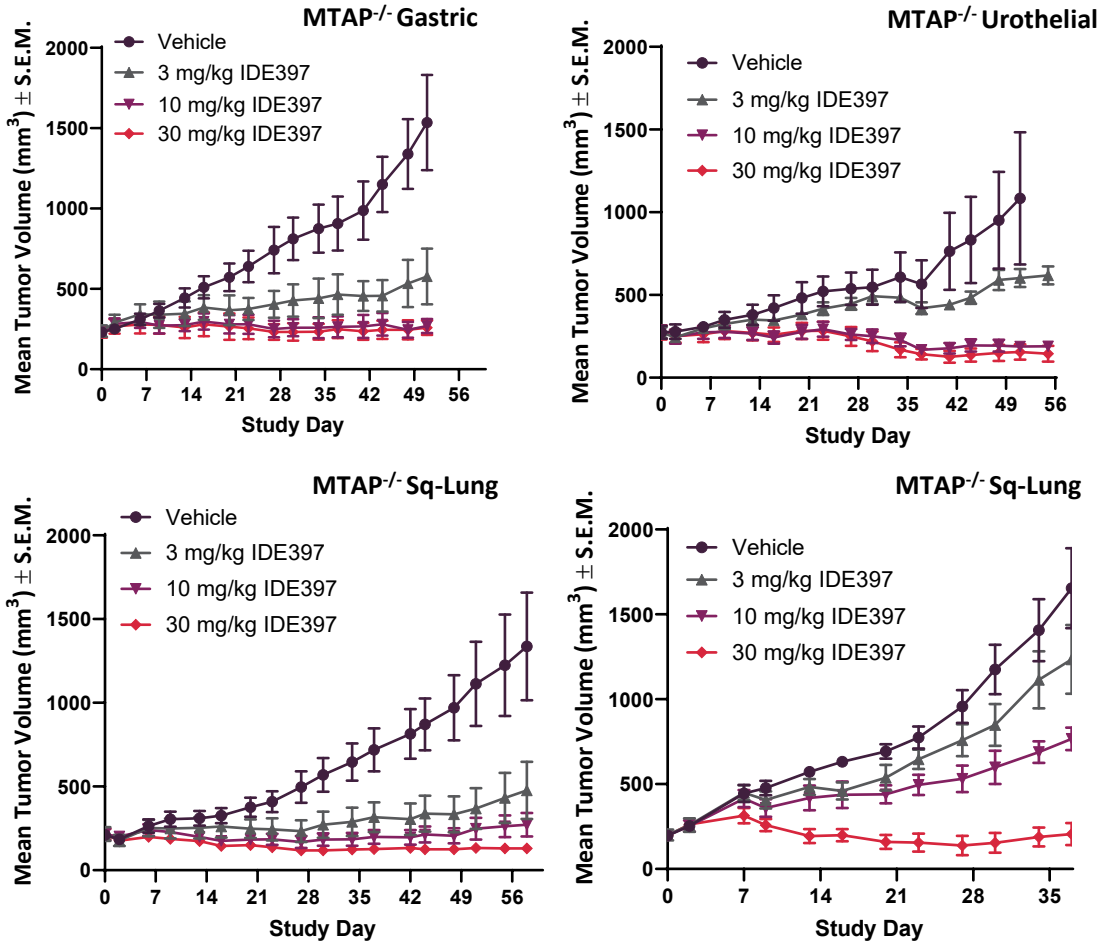
MAT2A

Strategies to address MTAP$^{-/-}$ Prevalence in ~15% of all Solid Tumors

MTAP-MAT2A Synthetic Lethality Biology



Robust monotherapy activity in lung, urothelial and gastric PDX



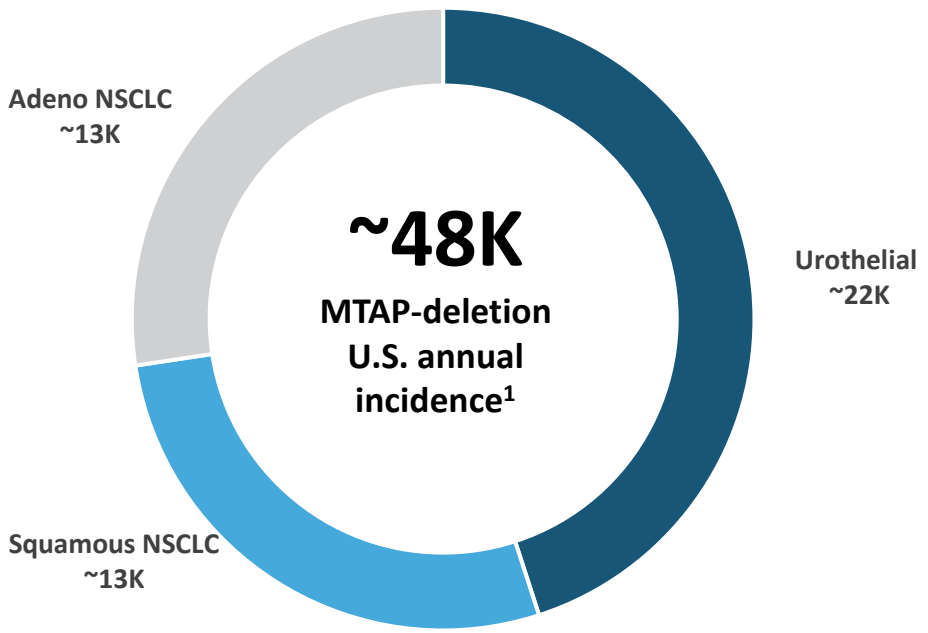
MAT2A = Methionine Adenosyltransferase 2a, MTAP = Methylthioadenosine Phosphorylase, MTA = Methylthioadenosine, PRMT5 = Protein Arginine Methyltransferase 5, PDX = Patient-Derived Xenograft

IDE397: Phase 2 Potential First-in-Class MAT2A Inhibitor

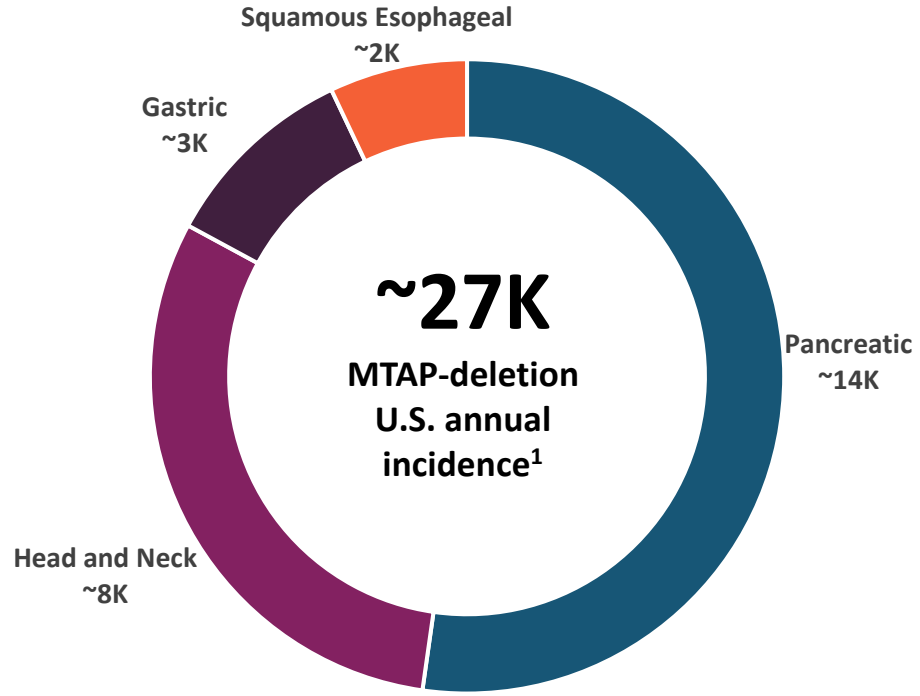
~48k U.S. Annual Incidence in MTAP-Deletion NSCLC and Urothelial Cancer

High Unmet Need: No FDA-Approved Therapies for MTAP-Deletion Solid Tumors

U.S. Annual Incidence in Priority Tumor Types



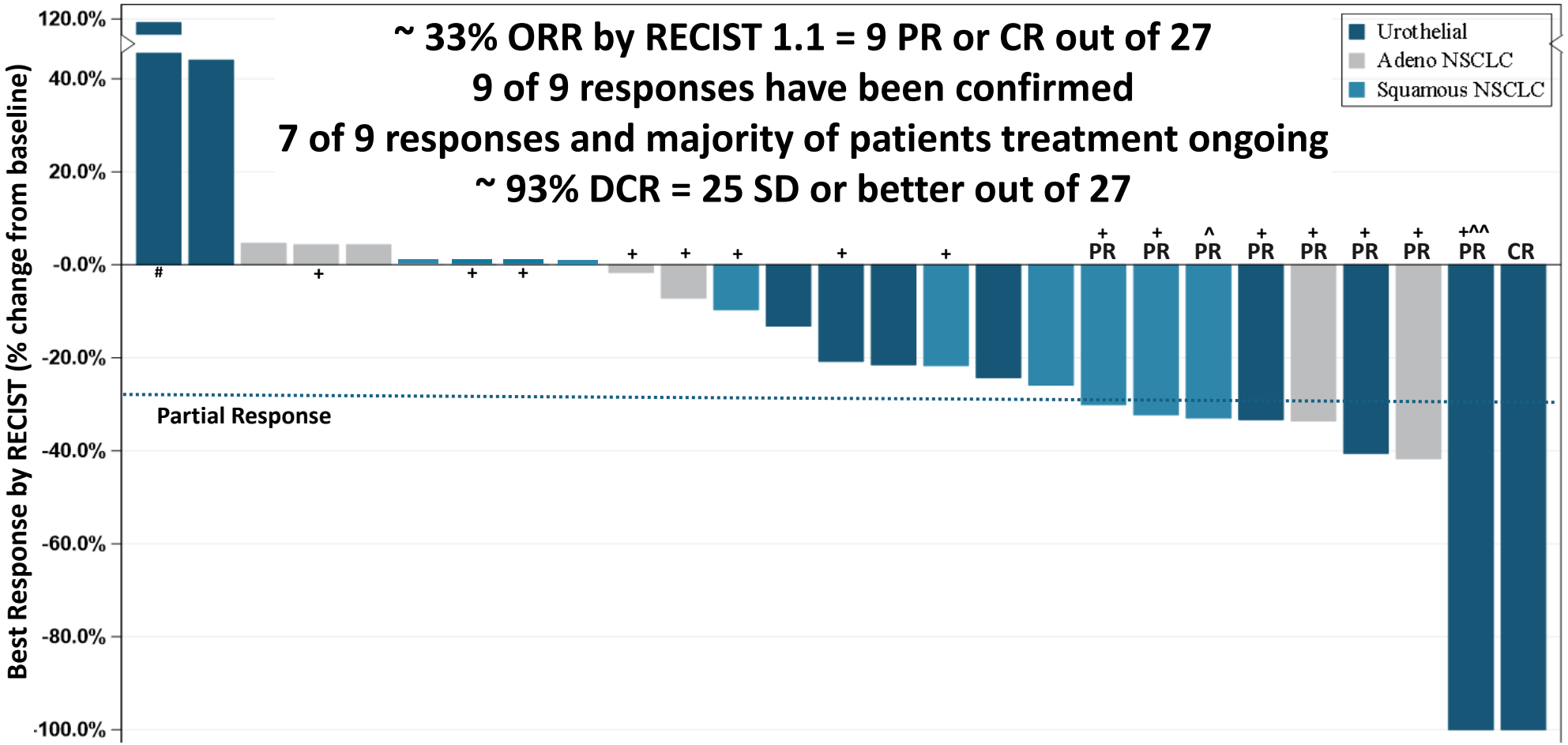
U.S. Annual Incidence in Potential Expansion Tumor Types



(1) Estimated addressable patient population based on SEER 2024 incidence and MTAP-deletion frequency from TCGA PanCancer Atlas, including frequency of 26% in urothelial, 19% in squamous NSCLC, 11% in adeno NSCLC, 21% pancreatic, 14% head and neck, 10% gastric, and 28% squamous esophageal cancers.
NSCLC = Non-Small Cell Lung Cancer

IDE397 Phase 1 Clinical Efficacy in MTAP-Deletion NSCLC & UC

Best Response by RECIST 1.1 at 30mg QD Phase 2 expansion dose[1]



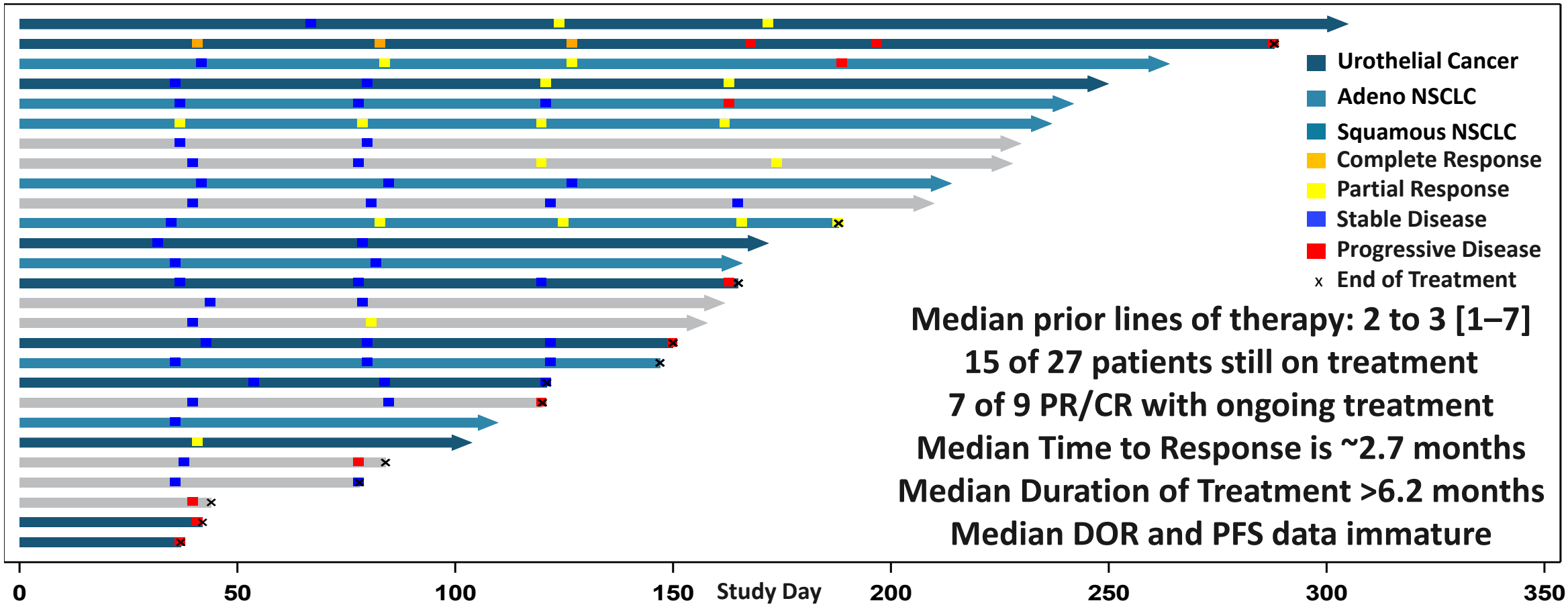
Efficacy by RECIST 1.1[1]	
Evaluable Pts	27
Best Response, n (%)	
CR	1 (4)
PR	8 (30)
SD	16 (59)
PD	2 (7)
ORR, n (%)	9 (33)
Confirmed, n^^	9
ORR, n (%), by Tumor (n)	
Squam NSCLC (8)	3 (38)
Adeno NSCLC (9)	2 (22)
Urothelial (10)	4 (40)
DCR, n (%)	25 (93)

(1) Evaluable Patients: Treated with ≥1 cycle (21 days) of IDE397 at the 30 mg expansion dose and with ≥1 post-baseline scan(s); # Patient received less than 75% of planned dosing prior to the first scan due to unrelated AEs in cycle 2; ^ Response evaluation by central review; ^^ PR with -100% best response had complete resolution of the target lesion; + patient still on treatment as of cut-off date. Data from an unlocked, unverified database as of 22AUG2024 data cut off; two patients confirmed response after the data cut. CR = Complete Response, PR = Partial Response, SD = Stable Disease, PD = Progressive Disease, ORR = Overall Response Rate, DCR = Disease Control Rate, c = Confirmed, NSCLC = Non-Small Cell Lung Cancer, UC = Urothelial Carcinoma, Squam = Squamous, Adeno = Adenocarcinoma, Pts = patients

IDE397 Phase 1 Clinical Efficacy in MTAP-Deletion NSCLC & UC

Time on treatment at 30mg QD Ph2 Expansion Dose

NSCLC & Urothelial Cancer Efficacy Evaluable Patients Treated at 30 mg (n=27)[1]



(1) Evaluable Patients: Treated with ≥1 cycle (21 days) of IDE397 at 30 mg expansion dose and with ≥1 post-baseline scan(s) Data from an unlocked, unverified database as of 22AUG2024 data cut off. The confirmed complete response urothelial patient progressed after the week 18 scan due to a drug-unrelated AE dose holiday and then restarted treatment. Two patients confirmed response after the data cut.

PFS = Progression Free Survival, DOR = Duration of Response

IDE397 Confirmed CR by RECIST 1.1 in UC Patient With MTAP-Deletion

Case Report and CT-Scan Images

Baseline Characteristics:

60+ years old male urothelial carcinoma

Treatment History:

- Neo-adjuvant cisplatin/gemcitabine
- Left nephro-ureterectomy
- Adjuvant Nivolumab

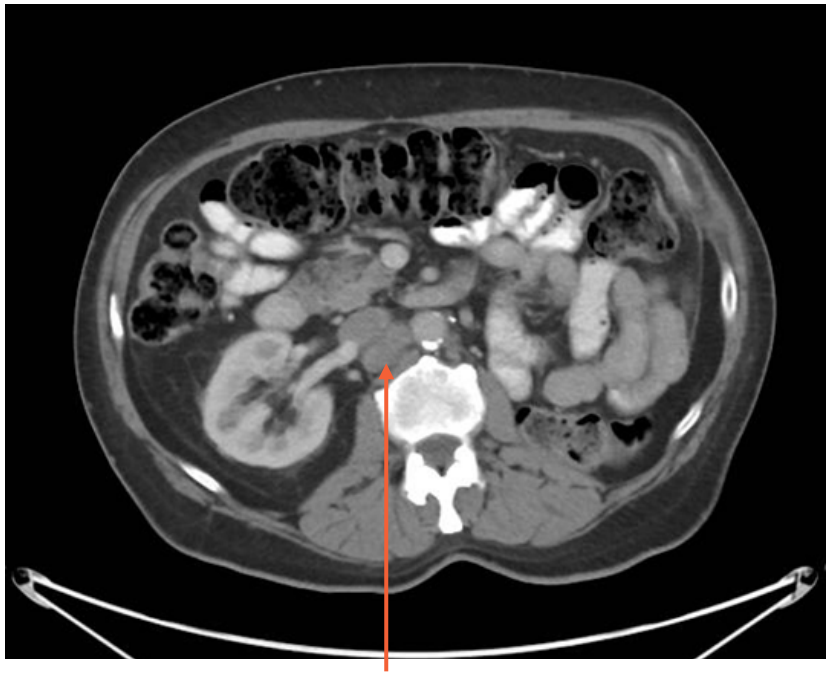
Recurrent disease while on adjuvant immunotherapy

RECIST 1.1 Evaluation:

CR by RECIST 1.1 at week 6 and confirmed at week 12

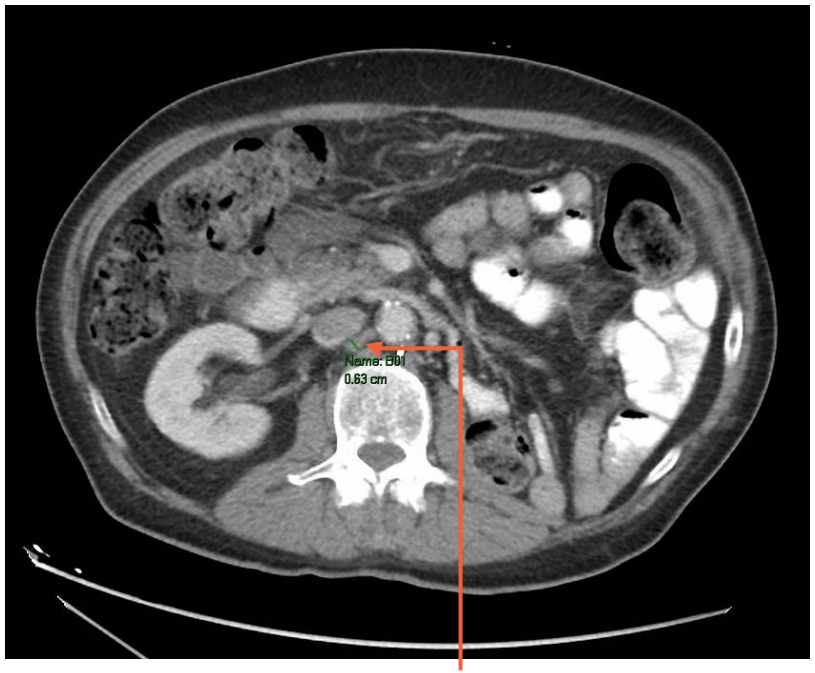
Urothelial Carcinoma with MTAP-Deletion: Maintained CR at Week 18

Baseline



Enlarged Retrocaval Lymph Node, 1.5 cm short axis

Week 18



Maintained Complete Response at week 18 scan

CR = Complete Response. IDEAYA Data (based on preliminary analysis of investigator provided images); tumor lesion reductions by investigator review, from an unlocked unverified database

IDE397 + Sacituzumab Govitecan Confirmed PR by RECIST 1.1 in Urothelial with MTAP-Deletion and FGFR3-TACC3 Fusion

Case Report and CT-Scan Images

Baseline Characteristics:

60+ years old male with Urothelial Cancer and MTAP-deletion and FGFR-TACC3 fusion

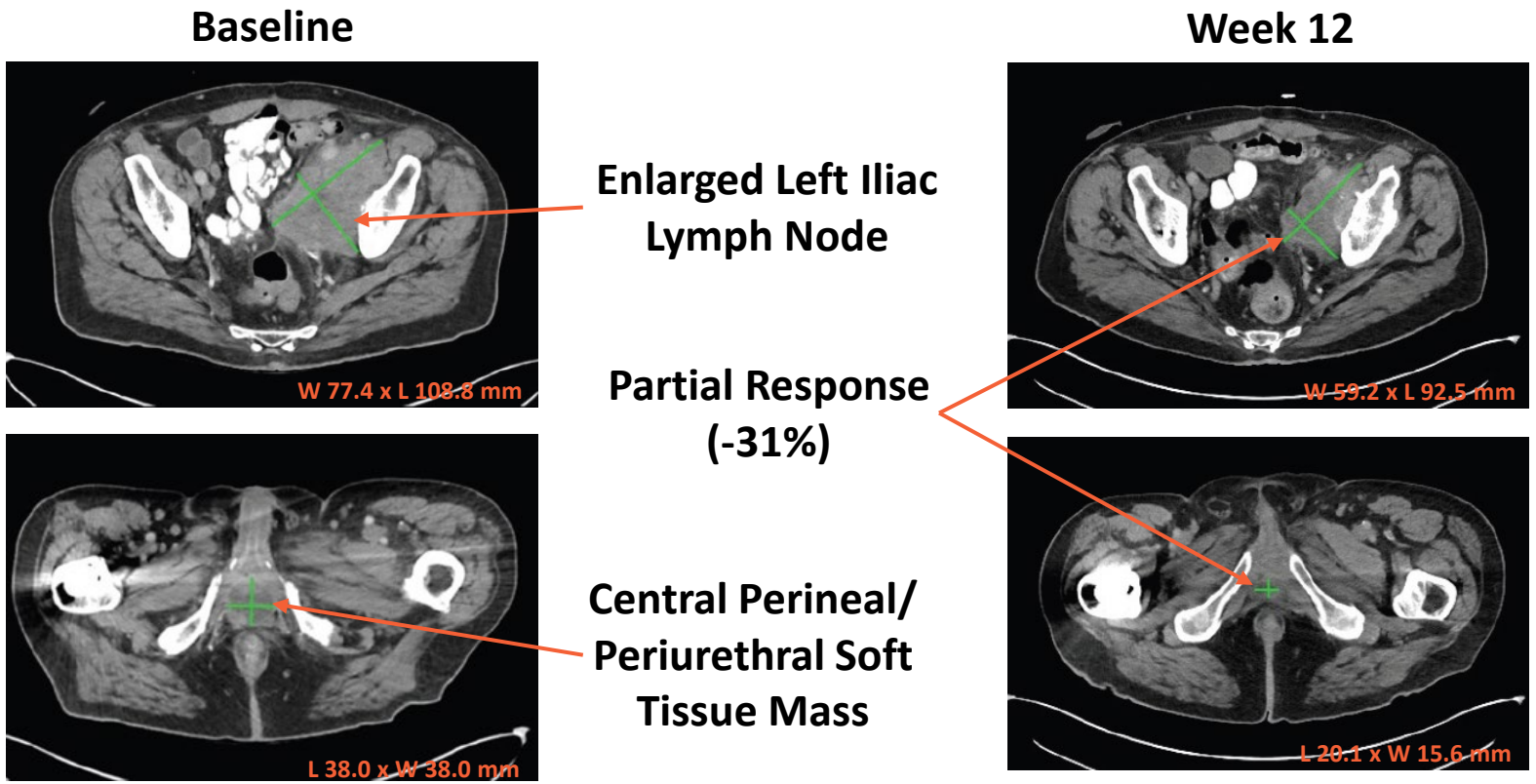
Treatment History:

- Transurethral resection
- Best response of PD to Enfortumab Vedotin (EV) + Pembrolizumab, and Erdafitinib

Clinical Evaluation:

PR by RECIST 1.1 at week 12, and confirmation at next scan with treatment ongoing

Urothelial Cancer MTAP-Deletion Patient: PR at Week 12 CT-Scan

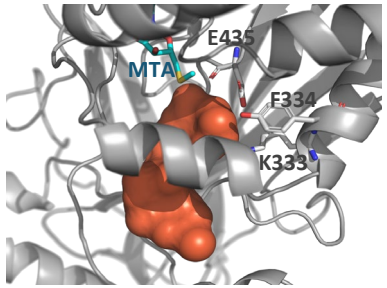


PR = Partial Response, PD = Progressive Disease. IDEAYA Data (based on preliminary analysis of investigator provided images); tumor lesion reductions by investigator review, from an unlocked unverified database.

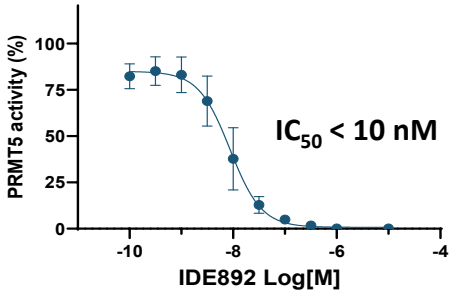
IDE892 DC: Potential Best-in-Class MTA-Cooperative PRMT5 Inhibitor

Target to Enable Wholly-Owned Clinical Combo with IDE397/MAT2A in H2 2025[1]

MTA-templated target binding

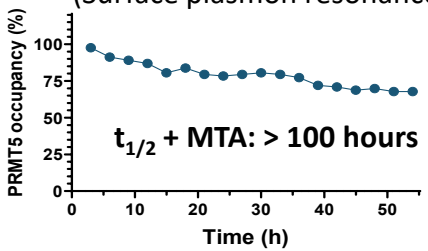


Potent biochemical inhibition

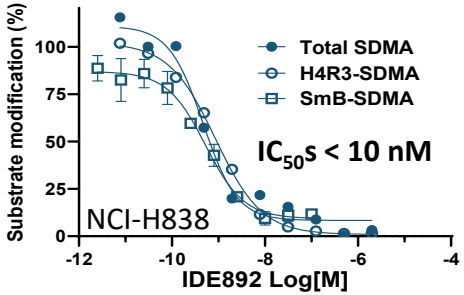


Persistent MTA-dependent target occupancy

(Surface plasmon resonance)

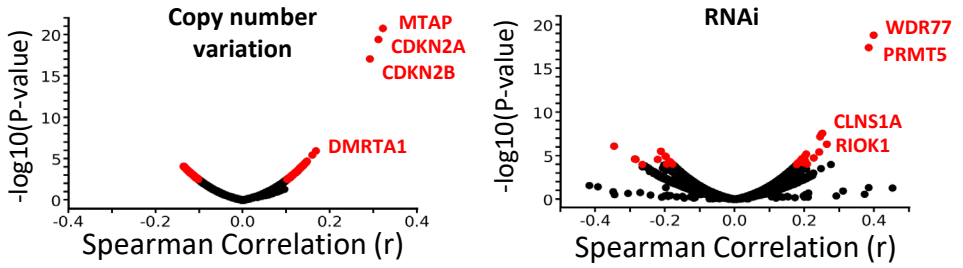


Robust pathway modulation



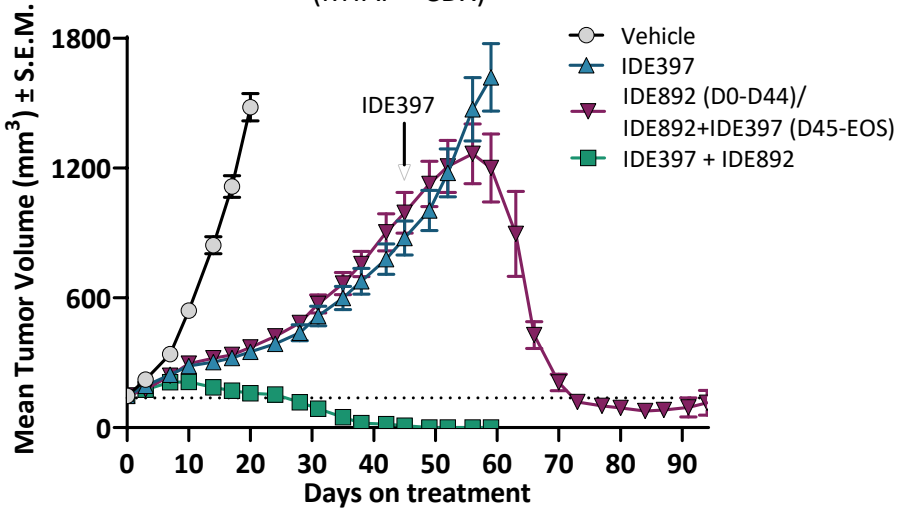
MTAP$^{-/-}$-specific cell killing

Correlation of Cell Features and IDE892 AUC across >800 cancer cell lines



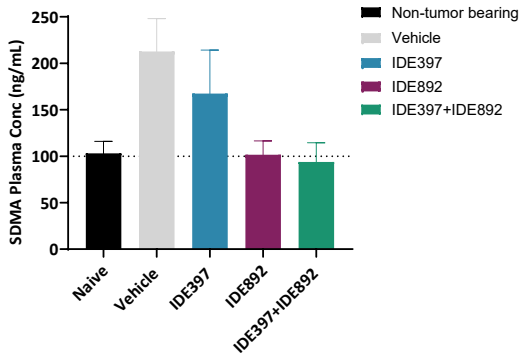
Exceptional IDE397 combination benefit

(MTAP$^{-/-}$ CDX)



Pathway sparing in normal tissue

Peripheral SDMA

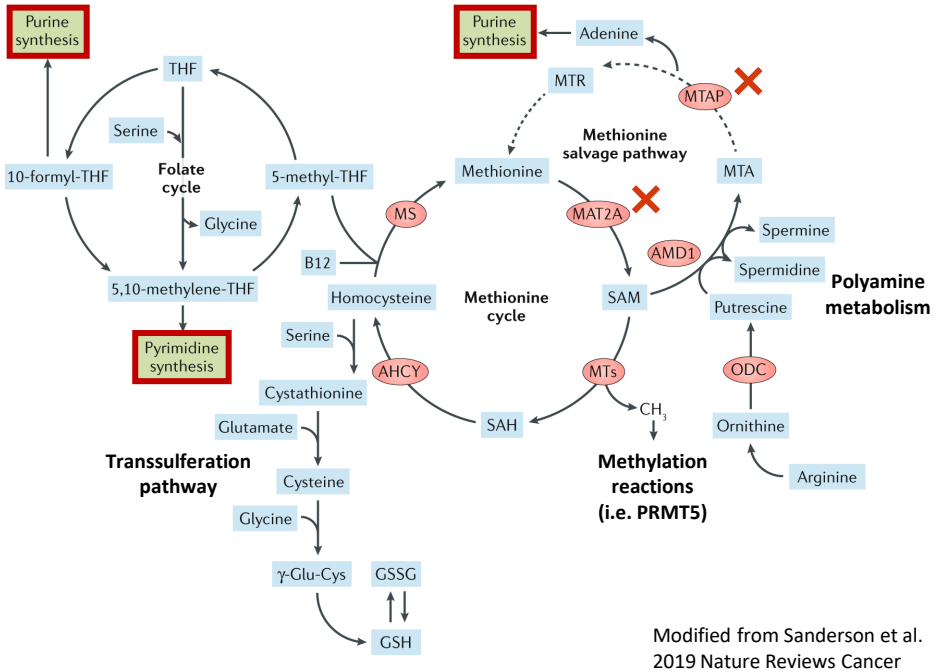


(1) IND-enabling studies ongoing with IND-filing targeted mid-year 2025. DC = Development Candidate

TRODELVY® + IDE397 Combination in MTAP-Deletion Urothelial Cancer

Disordered methionine metabolism underpins IDE397 combination opportunity with TOP1i-based ADC

IDE397 perturbs both nucleotide pools and mRNA splicing in MTAP$^{-/-}$ cells

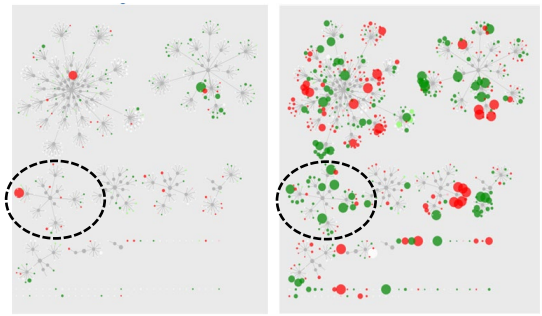


Modified from Sanderson et al. 2019 Nature Reviews Cancer

Metabolic perturbation by IDE397 selectively interacts with MTAP

Metabolite Cytoscape

Global (untargeted) metabolic profiling of MTAP^{wt} vs MTAP$^{-/-}$ +/- IDE397

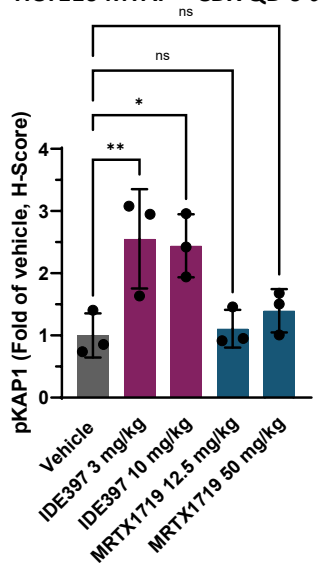


MTAP WT +/- IDE397 **MTAP$^{-/-}$ +/- IDE397**

Ovals indicate nucleotide subcluster (purine/pyrimidine); green-decrease, red-increase FDR< 0.05

IDE397 provokes DDR response in vivo

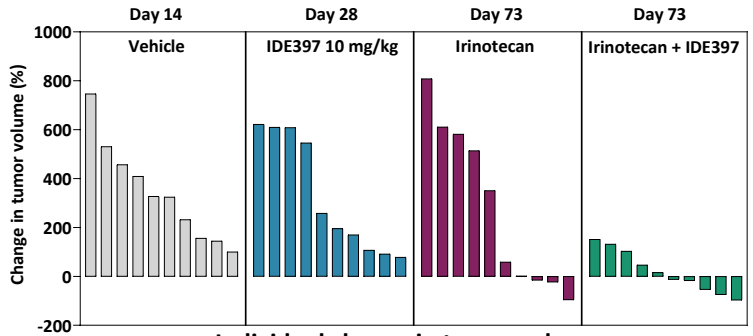
HCT116 MTAP$^{-/-}$ CDX QD 6 days



Key clinical correlates underscore combination opportunity

- MTAP$^{-/-}$ UC: shorter survival, lower RR to CPI, shorter PFS and OS with Enfortumab-Vedotin (EV)
- MTAP$^{-/-}$ status is associated with pemetrexed antitumor activity in UC
- A small study evaluating Trodelvy activity in UC post-EV suggested preferential activity in MTAP$^{-/-}$ tumors (RR 50% vs. 19% post EV)
- IDE397 demonstrated monotherapy efficacy in MTAP$^{-/-}$ UC (CR observed in a patient with short DFI post platinum and CPI, other tumor reductions and molecular responses seen)

TOP1i combination delivers durable benefit in challenging RT112/84 UC CDX model



Individual change in tumor volume

Individual tumors measured on day of study group termination as indicated; termination timing was based on endpoint criteria for tumor volume

ADC = Antibody Drug Conjugate, TOP1i = Topoisomerase I inhibitor, UC = Urothelial Carcinoma, RR = Response Rate, CPI = Check Point Inhibitor, PFS = Progression Free Survival, OS = Overall Survival, CR = Complete Response, DFI = Disease Free Interval , FDR = False Discovery Rate, DDR = DNA Damage Response, CDX = Cell Line-Derived Xenograft

IDE397 Phase 1/2 Clinical Development Plan in MTAP-Deletion Solid Tumors

Clinical Strategic Focus on High Conviction Rational Combinations

IDE397 – Clinical Profile

Exposure-Dependent Pharmacokinetic (PK) Profile with low C_{max}:C_{min}

Robust Pharmacodynamic (PD) Response observed

Monotherapy Expansion demonstrated clinical efficacy with Responses in Multiple High-Priority Tumor Types in Dose Expansion, including a Complete Response

IDE397 is strategically well positioned to evaluate both monotherapy and clinical combinations in MTAP-deletion solid tumors

IDE397 Monotherapy Expansion in Select MTAP-Deletion Solid Tumor Types

- IDE397 RDE Monotherapy Expansion: Squamous NSCLC and Urothelial Cancer
- Focus or Additional Indication Expansion

Trop2-ADC Combination – Targeting Clinical PoC in MTAP-Deletion UC and NSCLC

GILEAD

- IDE397 + Trodelvy®[1]: Urothelial Cancer and NSCLC

IDE397 + Trodelvy®
Clinical data update in MTAP UC in 2025; expansion in NSCLC

PRMT5 Combination – Targeting Clinical PoC in MTAP-Deletion NSCLC

- IDE397 + MTA-Cooperative PRMT5i[2]
- Other Potential Indications and/or Combinations

IDE397 + PRMT5
Enable wholly-owned clinical combo with IDE892 in H2 2025

(1) Trodelvy® = Gilead's Trop-2 directed ADC
(2) UC = Urothelial Cancer
(3) IDE892, IDEAYA PRMT5 inhibitor in IND-enabling studies

IDEAYA BIOSCIENCES

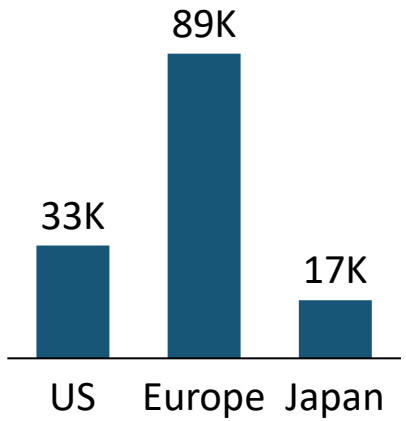
IDE849 (SHR-4849): Phase 1 DLL3 TOP1i ADC

First-in-Class Potential and Targeting Lineage Survival Oncogene Activity

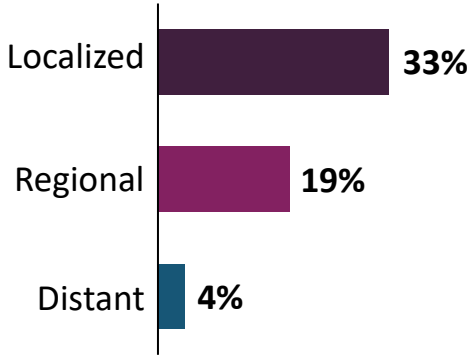
Significant unmet need in Small Cell Lung Cancer (SCLC)

Annual SCLC Patients[1] (2025)

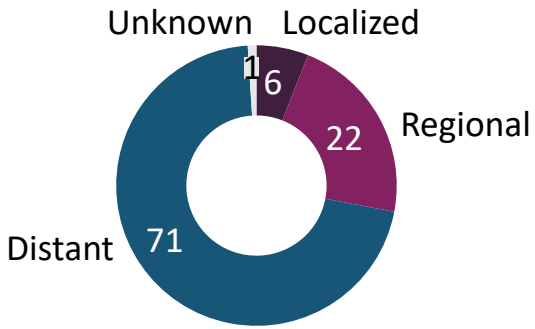
~15% of Lung Cancer (85% DLL3+)
(~350k Global Annual Incidence)



5 Year Survival



Stage at Diagnosis (%)

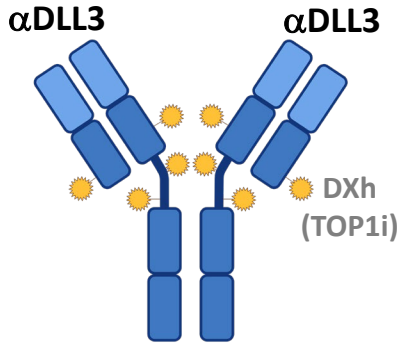


NETs

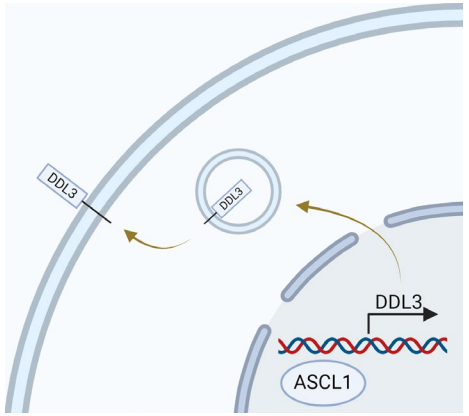
Neuroendocrine tumors (NET) secondary population with >100,000 patients[1] in US, Europe, Japan. 31-34% DLL3+

IDE849 (SHR-4849) potential first-in-class/best-in-class

The SCLC lineage survival oncogene, ASCL1, directly promotes DLL3 expression



SHR-4849/
IDE849



- DLL3 expression driven by the tumor-essential ASCL1 TF
- Humanized antibody with strong affinity and high selectivity
- Proprietary TOP1i payload (~4,000 patients treated)
- Internalization-dependent cleavable linker
- Optimized DAR value of 8
- High plasma stability
- 120X estimated therapeutic index

(1) Incidence plus newly recurrent.

Sources: WHO Globocan 2022, SEER, Rojo, F., at al., Lung Cancer. 2020;147:237–243; Tanaka, K., at al., Lung Cancer. 2018 Jan:115:116-120; Yao, J., at al., The Oncologist, 2022, 27, 940–951; Ali, G., at al., Front. Oncol. 11:729765; Song, H., at al., Exp Ther Med 16: 53-60, 2018, Ideaya Commercial Analysis, 2024 AACR. Abstract 3146/27

DLL3 = Delta-Like Ligand 3, ADC = Antibody Drug Conjugate, TOP1i = Topoisomerase I Inhibitor, DAR = Drug-to-Antibody Ratio, TF = Transcription Factor

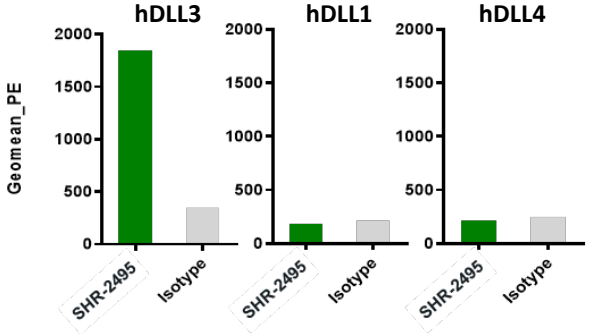
IDE849 (SHR-4849): Well-tolerated Robust Antitumor Activity in DLL3+ SCLC

DLL3-Specific Tumor Cell binding, Internalization and Payload Release

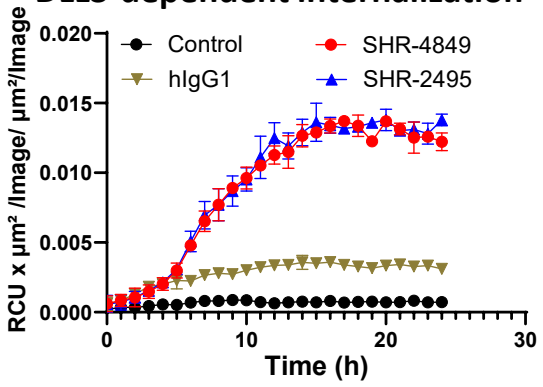
SHR-4849 displays DLL3-specific tumor cell binding, internalization and payload release

DLL3-specific binding

(SHR-2495: antibody component of IDE849)

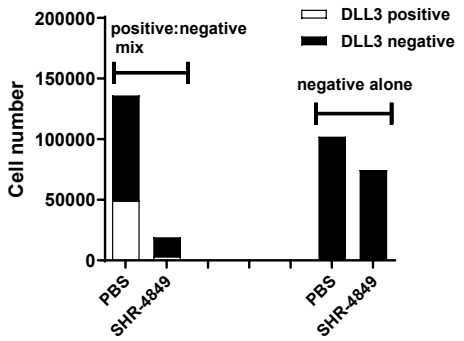


DLL3-dependent internalization



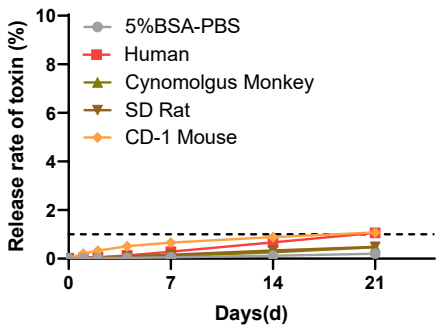
DLL3-specific cytotoxicity

Bystander Killing Assay



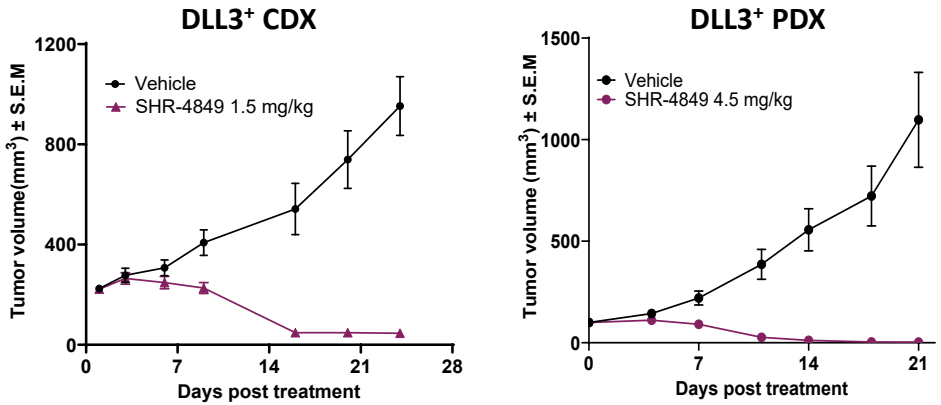
ADC serum stability

SHR-4849 (100 µg/mL)

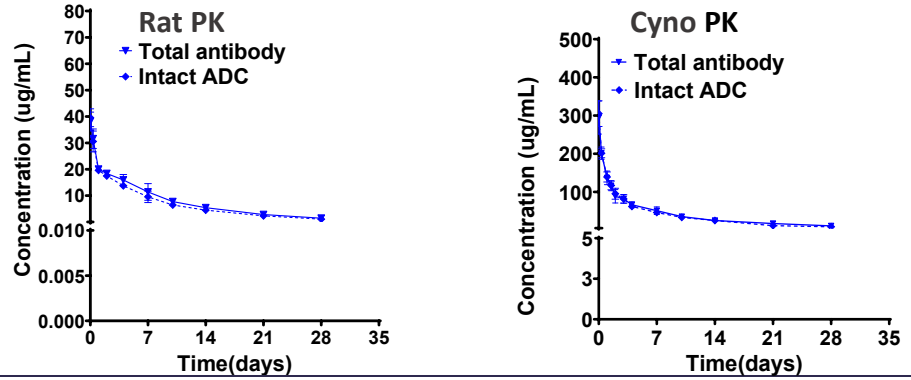


Deep regressions in DLL3+ CDX/PDX with exceptional linker/payload stability in circulation

Deep regressions observed in DLL3+ SCLC



Limited to no payload deconjugation in vivo



Source: Hengrui Pharma

CDX = Cell Line-Derived Xenograft, PDX = Patient-Derived Xenograft, PK = Pharmacokinetics

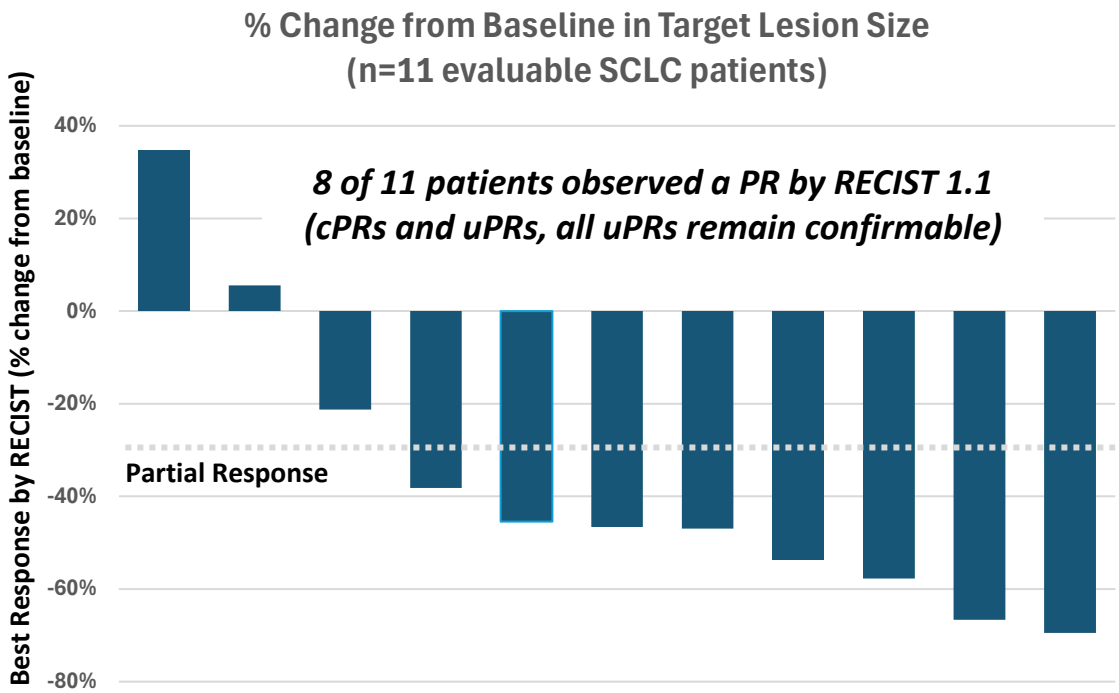
IDE849 (SHR-4849): Potential First-in-Class with Preliminary Ph1 Clinical PoC

Phase 1 FIH Study of DLL3 Topo-1-Payload ADC in Pre-Treated SCLC Patients

Phase 1 Dose Escalation in China in Pre-Treated SCLC Patients[1]

- **Preliminary Clinical PK Summary**
  - Dose dependent increase in exposure
  - Promising T-Ab to ADC ratio
- **Preliminary Clinical Efficacy Summary[2]**
  - 8 of 11 evaluable SCLC patients observed a partial response by RECIST 1.1, resulting in a ~73% ORR (confirmed and unconfirmed, all unconfirmed PRs remain confirmable)
- **Preliminary Clinical Safety Summary**
  - TRAEs were largely Grade 1 or 2
  - No AE leading to discontinuation (related or unrelated)
  - Maximum tolerated dose has not yet been reached
  - Most commonly observed TRAEs: white blood cell count decreased, anemia, neutrophil count decreased, nausea and platelet count decreased

Tumor Reductions and Responses seen in most evaluable subjects after IDE849 Treatment[1]



(1) All unconfirmed responses pending further evaluation
(2) Clinical efficacy summary at therapeutic dose levels
Source: Hengrui Pharma. Data Cut off Dec 10, 2024.
ADC = Antibody Drug Conjugate, SCLC = Small Cell Lung Cancer, T-Ab = Total Antibody, PR = Partial Response, u = Unconfirmed, c = Confirmed

IDE849 (SHR-4849): Potential First-in-Class with Preliminary Ph1 Clinical PoC

Pre-Treated SCLC Patient Case Study and Preliminary IDEAYA Clinical Development Plan

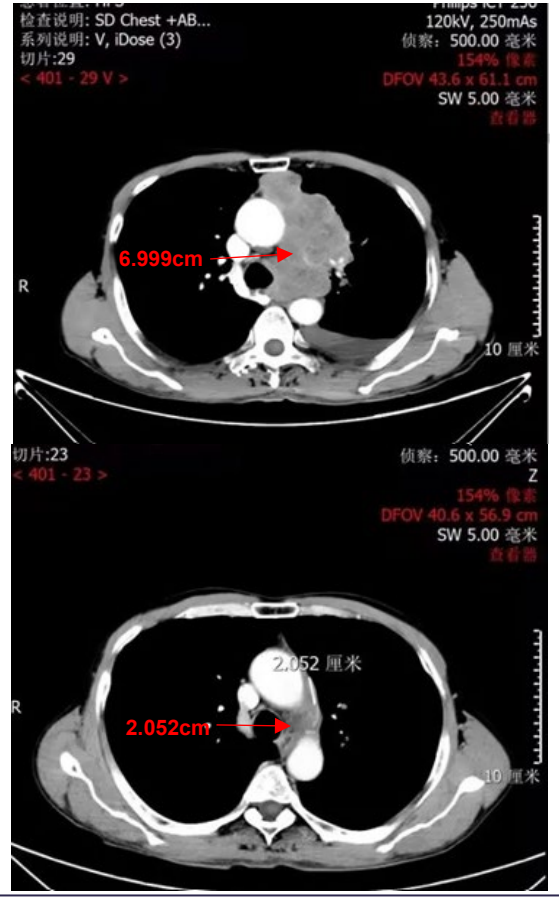
Case Example in Phase 1 FIH Dose Escalation

A 70-year-old male with extensive stage SCLC who had failed prior PD-L1 and platinum doublet treatment

The subject was treated with IDE849 and achieved PR at Week 6 with a 70.6% reduction in the large mediastinal tumor mass

Baseline

Week 6



IDE849 Phase 1/2 Clinical Development Plan

- IDE849 Monotherapy Dose Escalation and Expansion
  - Dose Escalation
  - Expansion Cohort: SCLC
  - Expansion Cohort: NETs
- IDE849 Combination with IDE161/PARG
  - IDE849 + IDE161: SCLC and NETs

Preliminary Clinical Strategy:

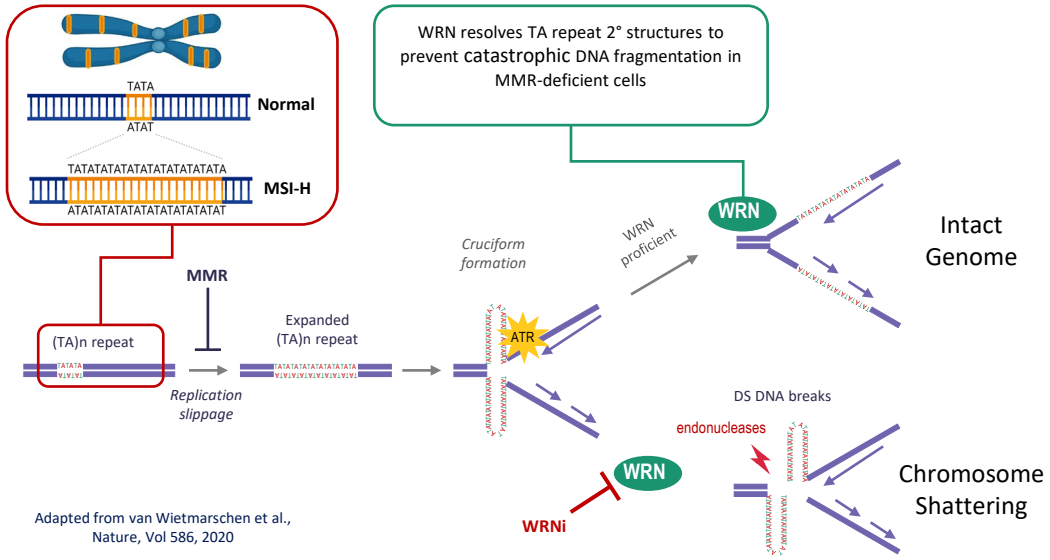
- Potential monotherapy path in 2L plus SCLC
- Evaluate clinical combinations, including with SOC, in 1L SCLC
- Evaluate NETs as monotherapy, including potential basket trial
- Target to enhance durability with IDE849 + IDE161/PARG combo

SOC = Standard of Care
Source: Hengrui Pharma

IDE275 (GSK959): Potential First-in-Class Ph1 Werner Helicase Inhibitor

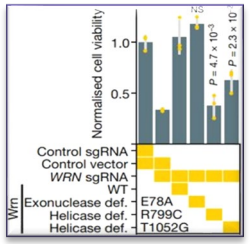
WRN Helicase Activity is Specifically Essential for Survival of MSI-high/dMMR Cancer Cells

WRNi: tumor-intrinsic DNA damage

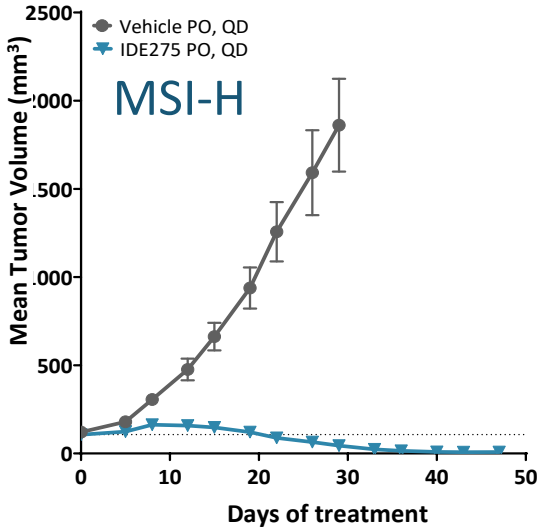


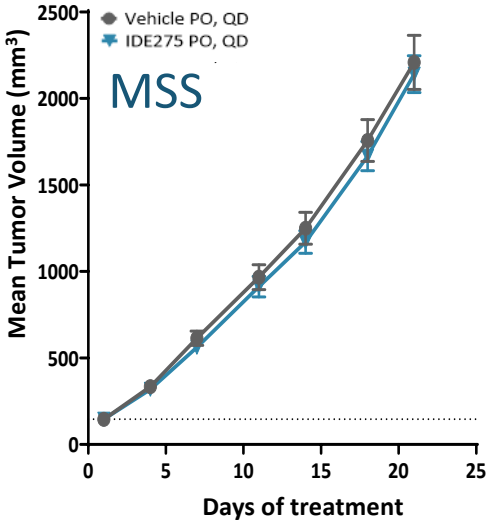
Adapted from van Wietmarschen et al., Nature, Vol 586, 2020

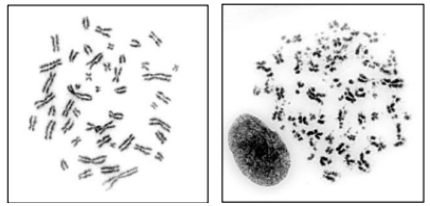
WRN helicase domain (not exonuclease domain) most critical as therapeutic target



Anti-tumor response is specific for MSI-H







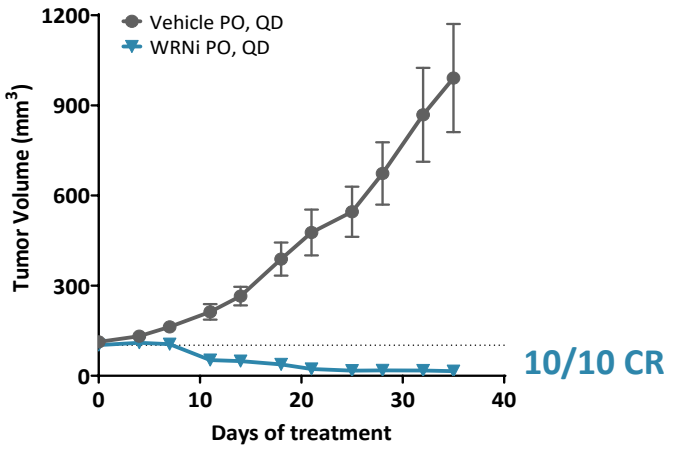
Untreated Treated

WRN = Werner Helicase, MSI = Microsatellite Instability, dMMR = Deficient Mismatch Repair, PO = Oral, QD = Once Daily

IDE275 (GSK959) Werner Helicase Inhibitor Demonstrates Robust Anti-Tumor Activity in MSI-H and Heavily Pre-Treated Tumors

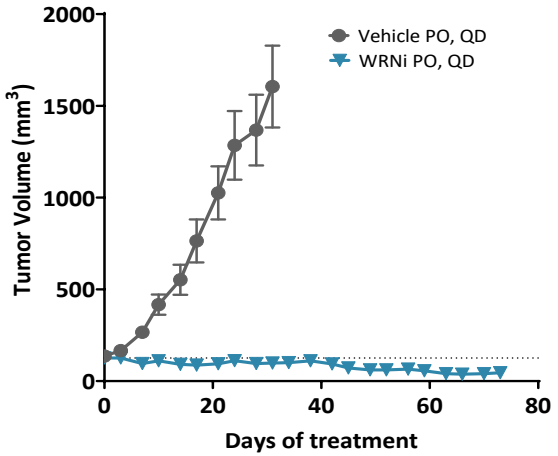
Supports Clinical Strategy to Expand beyond MSI-H Colorectal Cancers

Endometrial CDX



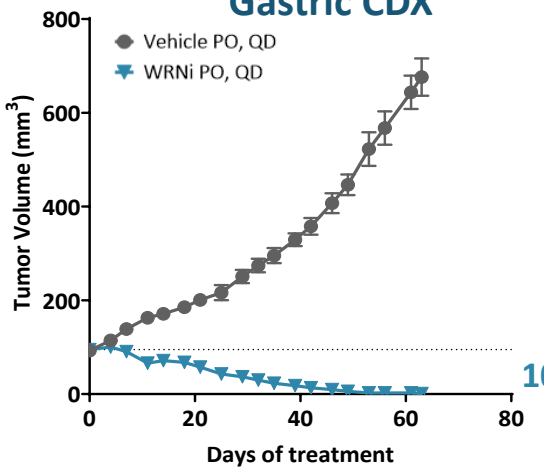
10/10 CR

Endometrial PDX



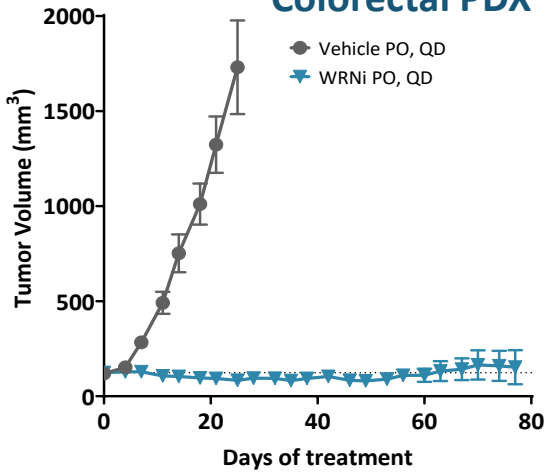
4/10 CR

Gastric CDX



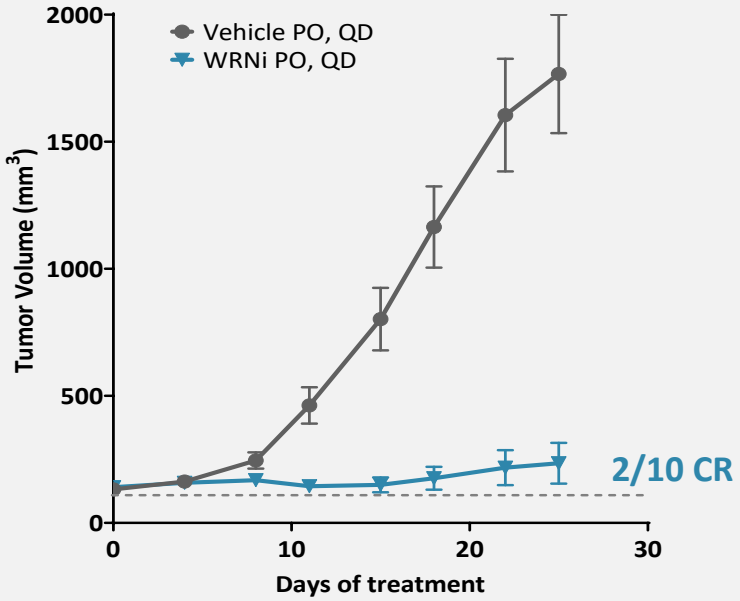
10/10 CR

Colorectal PDX



2/10 CR

Treatment-relapsed CRC PDX



2/10 CR

48 yo male treated with:

- 1L FOLFOX
- 2L XELIRI + bevacizumab
- 3L nivolumab

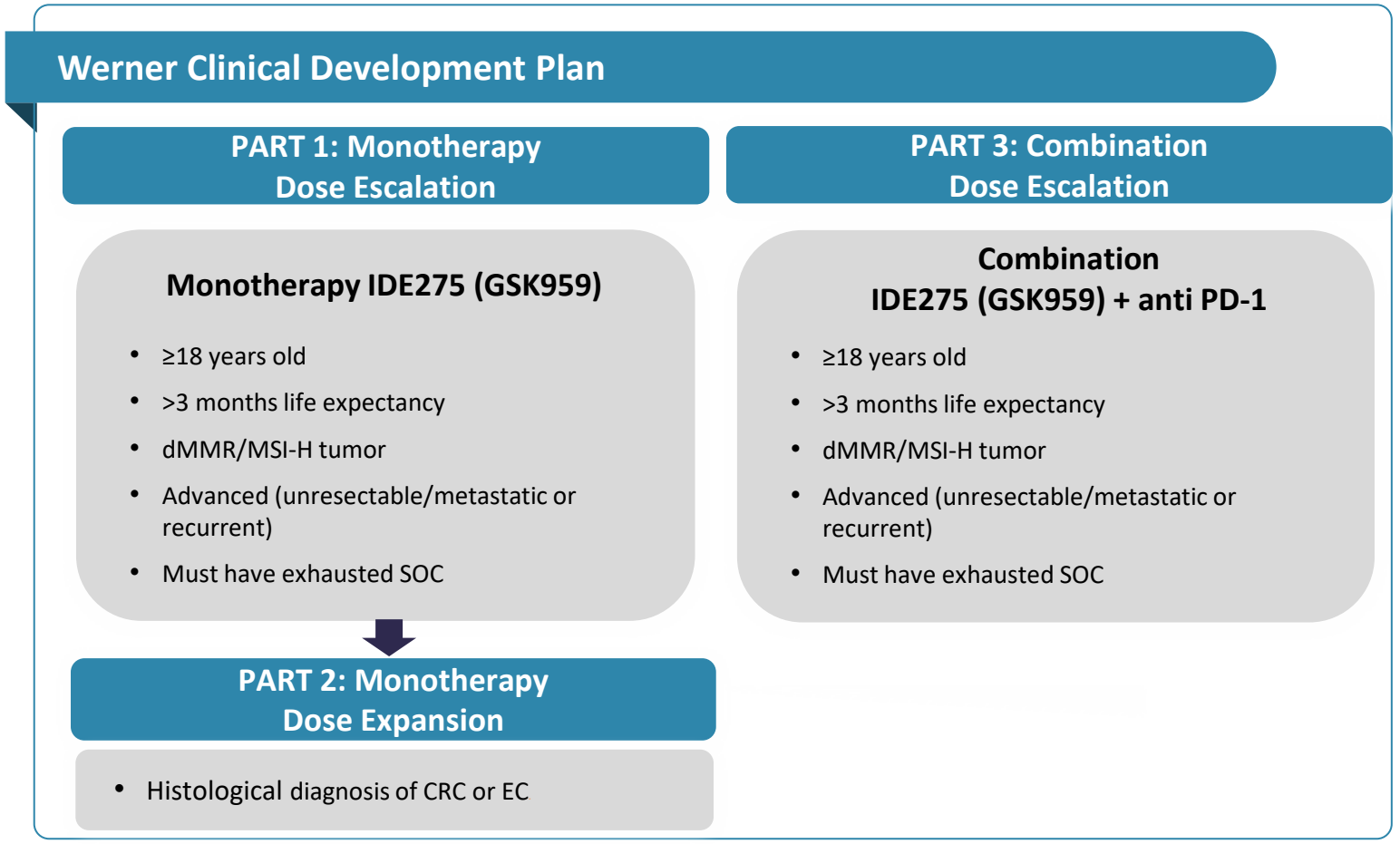
CDX = Cell Line-Derived Xenograft, PDX = Patient-Derived Xenograft, CR = Complete Response, CRC = Colorectal Cancer

IDE275 (GSK959): Phase 1 Werner Helicase Inhibitor

Clinical Development Plan

IDE275 (GSK959) Werner Helicase Inhibitor

- IDE275 (GSK959) has demonstrated robust and selective synthetic lethality preclinically in the high microsatellite instability (MSI-High) biomarker setting
- Phase 1 clinical trial enrolling patients having tumors characterized by MSI-High (NCT06710847)



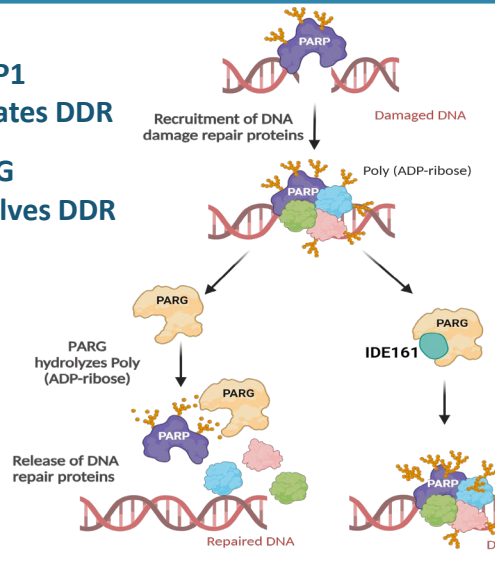
GSK Strategic Partnership: 50/50% US Profit Share and ex-US Royalties, ~$1B Milestones, incl. up to $20M Preclinical / Ph1 Clinical; Cost Share 80% (GSK) / 20% (IDEAYA); Potential Combination with GSK's Jemperli™, a PD-1 IO Agent

CRC = Colorectal Cancer, EC = Endometrial Cancer, IO = Immunotherapy, SOC = Standard of Care

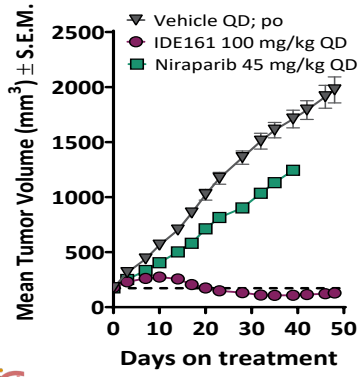
IDE161: Potential First-in-Class Phase 1 PARG Inhibitor

PARG Inhibition is synthetic-lethal with replication stress

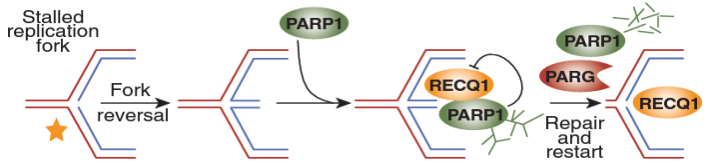
- PARP1 initiates DDR
- PARG resolves DDR



- PARG inhibition promotes death by mitotic catastrophe



Oncogene-induced replication stress confers PARG dependency

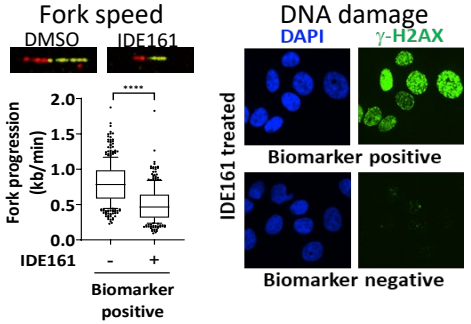


Adapted from Pillay et al. Cancer Cell , 2019

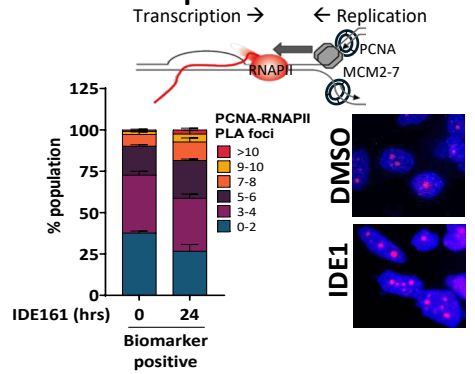
- Replication stress promotes replication fork reversal
- PARG supports replication fork restart

IDE161 disrupts DNA replication fork fidelity

IDE161 induces fork progression defects leading to pan-nuclear DNA damage

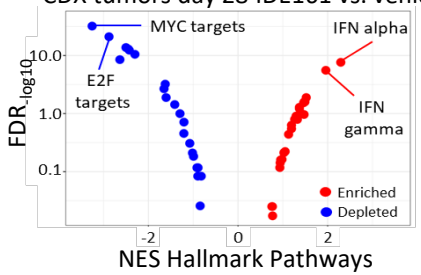


IDE161 induces transcription-replication conflict



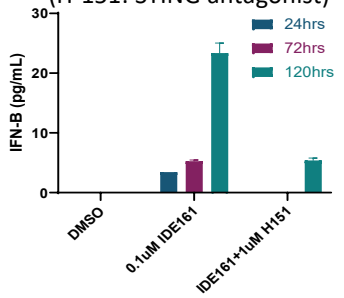
IDE161 activates innate-immune response in tumors *in vivo*

CDX tumors day 28 IDE161 vs. vehicle



IDE161 induces STING-dependent IFN in tumor cells in culture

(H-151: STING antagonist)



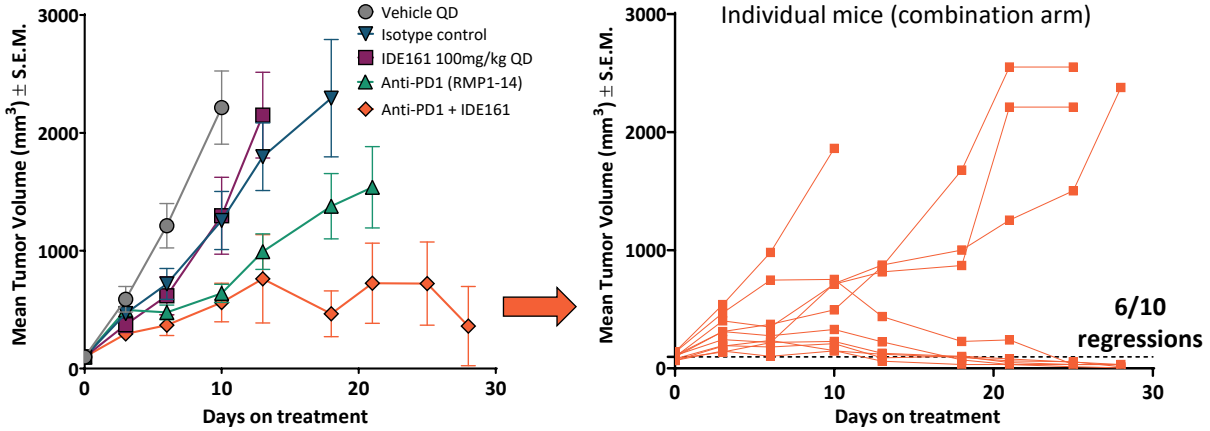
PARG = Poly (ADP-ribose) Glycohydrolase, DDR = DNA Damage Response, FDR = False Discovery Rate, IFN = Interferon

IDE161 Combination Strategies with PD-1 and TOP1i-ADCs

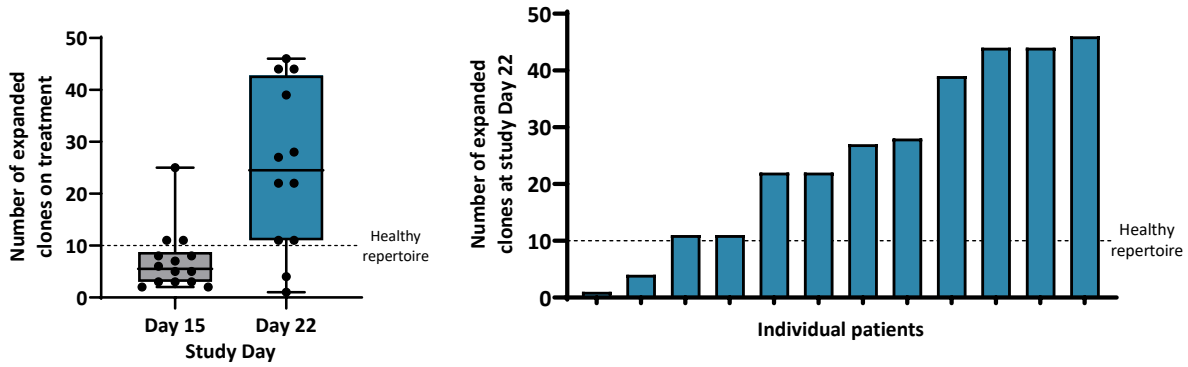
High Conviction Mechanistic Rationale with Potentially Broad Development Opportunity

STING activation by IDE161 enhances antitumor immunity

Anti-PD-1 combination benefit associated with enhanced immune cell tumor infiltration

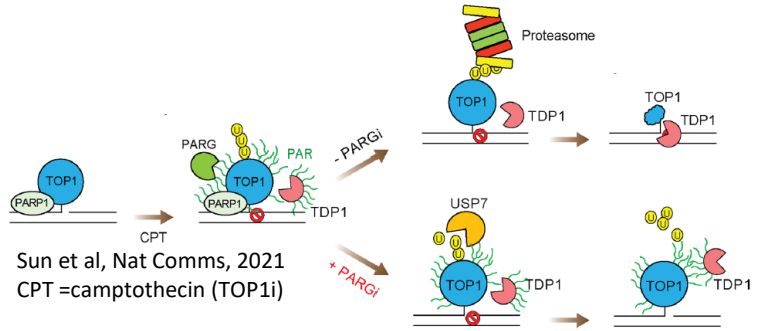


T cell receptor sequencing of clinical PBMC specimens provides evidence of adaptive immune response following IDE161 monotherapy

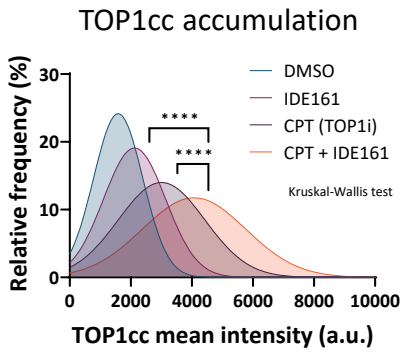


IDE161/TOP1i DDR interaction enhances ADC efficacy

Dual inhibition of TOP1 & PARG induces unresolvable DNA-protein crosslinks

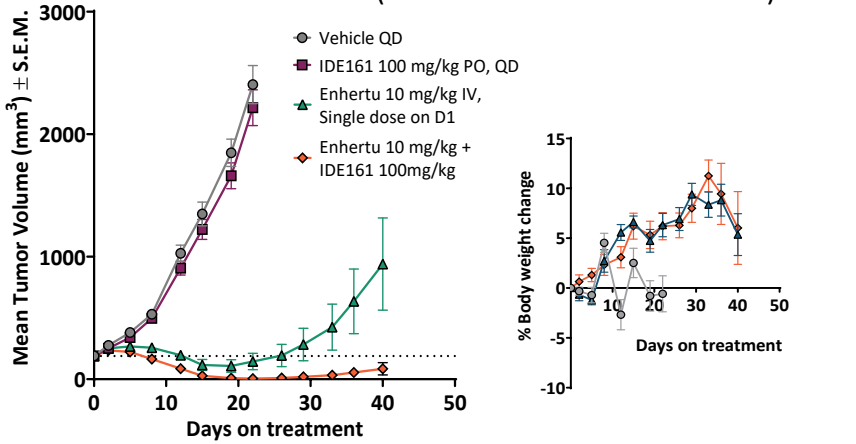


Sun et al, Nat Comms, 2021
CPT =camptothecin (TOP1i)



Potential for IDE161 as platform / backbone combo partner for TOP1i-ADCs

HER2⁺ lung; 60% complete responses in combination (Fam-trastuzumab deruxtecan)



PPMC = Peripheral Blood Mononuclear Cells, TOP1i = Topoisomerase I Inhibitor, ADC = Antibody Drug Conjugate

IDE161 Phase 1/2 Clinical Development Plan in Solid Tumors

Clinical Strategic Focus on Rational Combinations with TOP1i-ADCs and PD-1

IDE161 Phase 1/2 – Monotherapy and Combination Clinical Development Plan

IDE161 Monotherapy Dose Escalation and Expansion in HRD Solid Tumors[1]

- Dose Escalation
- Expansion Cohort: Priority Tumor Type

IDE161 + KEYTRUDA® (pembrolizumab) in Endometrial Cancer

- MERCK (2) — IDE161+ KEYTRUDA in Endometrial Cancer – Targeting 2025 clinical expansion

IDE161 Topo ADC Combination Opportunities Validated Preclinically

- HENGRUI (3) — IDE161+ IDE849 (DLL3 ADC)
- BIOCYTOGEN (4) — IDE161+ IDE034 (B7H3/PTK7 Bispecific Ab-TOP1i ADC DC)

Activity in PARPi- and Platinum-Resistant Settings	Differentiated Sensitivity relative to PARPi's	Targeting Improved Safety Profile relative to PARPi's

IDE161 monotherapy expansion initiated in priority tumor type

IDE161 + Keytruda clinical combo FPI achieved

Targeting to enable IDE161 + TOP1i-ADC clinical combinations in 2025

Facile peripheral PD Biomarker for PARGi based on measurement of PAR in blood samples (PBMC's)

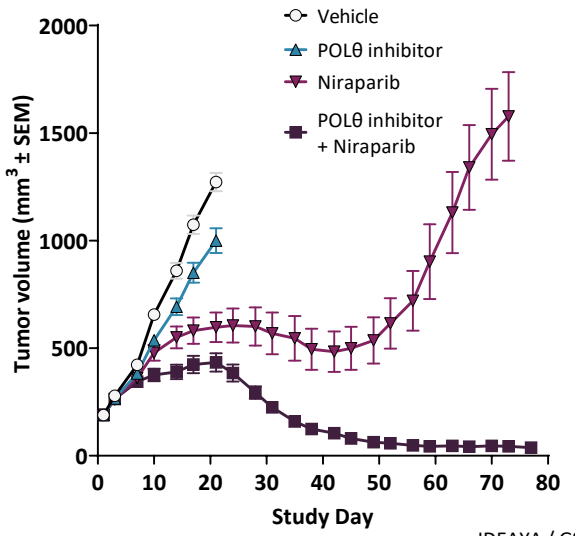
(1) Clinicaltrials.gov: NCT05787587
(2) Pursuant to Merck Clinical Trial Collaboration and Supply Agreement for IDE161 + Keytruda®, Merck's anti-PD-1 therapy; the Company will sponsor the study and Merck will provide Keytruda at no cost
(3) Pursuant to exclusive license agreement with Jiangsu Hengrui Pharmaceuticals Co., Ltd for worldwide rights outside of Greater China
(4) Pursuant to exclusive worldwide licensing and option agreement with Biocytogen
PARG = poly (ADP-ribose) glycohyrdolase, PAR = poly (ADP-ribose), PBMC = Peripheral Blood Mononuclear Cells, PSA = Prostate Specific Antigen, EC = Endometrial Cancer, CRC = Colorectal Cancer

IDE705 (GSK101): Potential First-in-Class Ph1 Pol Theta Helicase Inhibitor

Phase 1 in Combination with Niraparib (PARPi)

Pol Theta Helicase *In Vivo* Activity

IDE705(GSK101) + PARPi

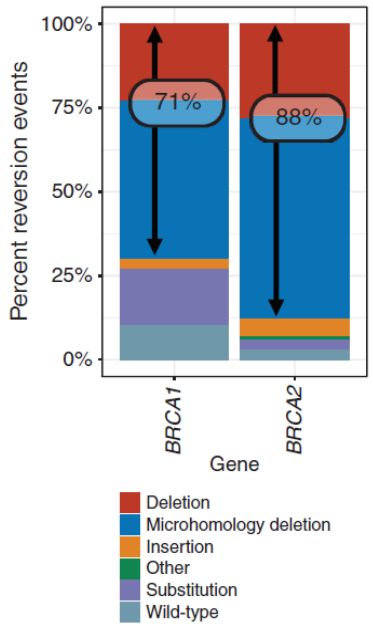


IDEAYA / GSK Data

Observed Deep and Durable Responses in Multiple Xenograft Models

BRCA 1/2 Clinical Reversions

BRCA Reversions Mediated by MMEJ



Cancer Res. 2020, DOI: 10.1158/2159-8290

Clinical Development Strategy

Pol Theta Helicase Inhibitor + PARP Inhibitor

Pol Theta Helicase Inhibitors Disrupt MMEJ Alternative DNA Damage Repair:

- Inhibit DSB Repair by MMEJ
- Dysregulate Replication Fork Stabilization

Potentiate PARPi Efficacy	Prevent PARPi Resistance	Overcome PARPi Resistance

Potential Clinical Opportunities

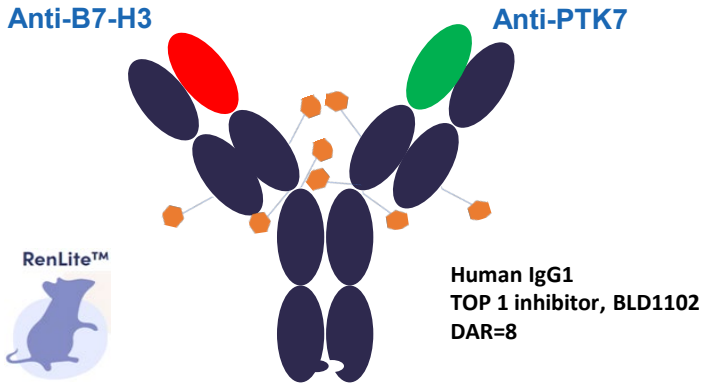
GSK Strategic Partnership: Global Royalties with GSK covering all Costs, ~$1B Milestones, incl. up to $20M
Preclinical / Ph1 Clinical Potential Combination with GSK's Zejula™, a PARP Inhibitor

PARP = Poly (ADP-ribose) Polymerase, MMEJ = Microhomology-Mediated End Joining, DSB = Double Strand Break

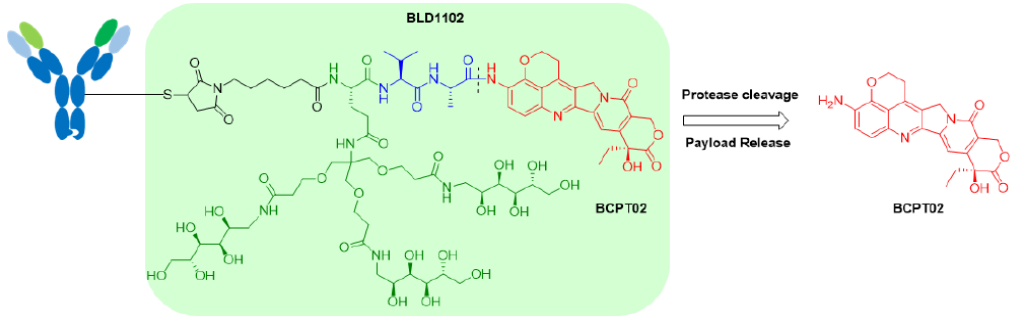
IDE034 DC: Potential First-in-Class B7H3/PTK7 TOP1i Bi-Specific ADC[1]

Dual Tumor-Associated Antigen Targeting for Potential Enhanced Therapeutic Window

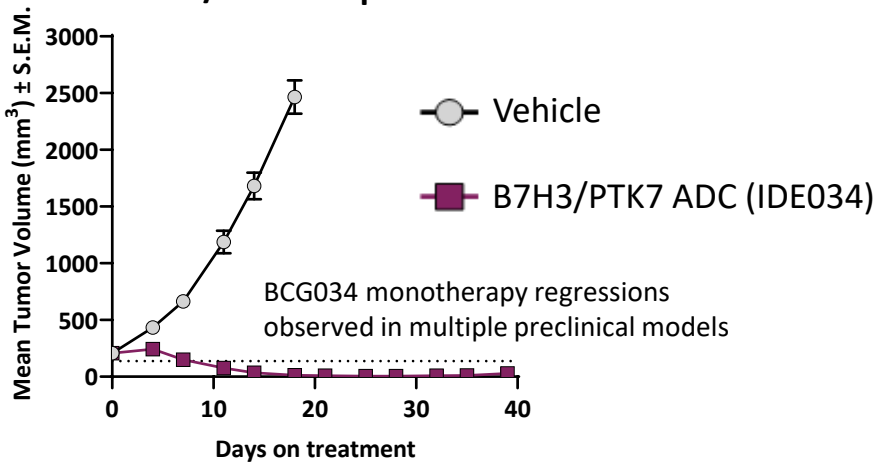
IDE034: B7H3/PTK7 Bispecific Ab-TOP1i ADC[1]



Proprietary Topoisomerase I Linker-Payload



B7H3+/PTK7+ Expression PDX Model



- Enhanced tumor versus normal cell binding
- Enhanced internalization efficiency
- Meaningful double-positive disease population[2]

Indication	B7H3/PTK7 Double Positive %
Lung	29.8%
Colorectal[3]	45.9%
HNSCC	27.1%
Ovarian	23.1%

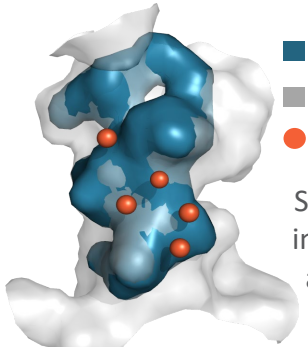
Substantial addressable B7H3/PTK7 patient population

(1) IDE034: B7H3/PTK7 Top1i Bispecific ADC development candidate (DC). Exclusive worldwide licensing and option agreement with Biocytogen; IND-enabling studies ongoing with IND-filing targeted in H2 2025
(2) IDEAYA analysis of Human Protein Atlas
(3) Human Protein Atlas annotates colorectal cancer as bowel cancer
DAR = Drug Antibody Ratio, IND = Investigational New Drug

IDE251 DC: Dual KAT6/7 Inhibitor with High Selectivity vs KAT Family[1]

Potent Pathway Modulation Delivers Robust Biomarker-Specific Single-Agent Activity

IDE251 solves considerable design challenge

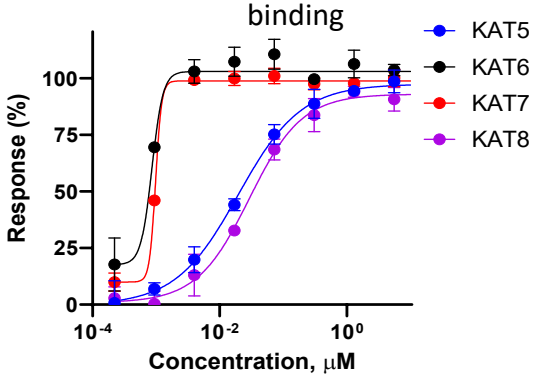


KAT7 pocket (270 Å^3)
KAT6 pocket (614 Å^3)
Residue differences

Substantial difference in binding site volume and residue identity

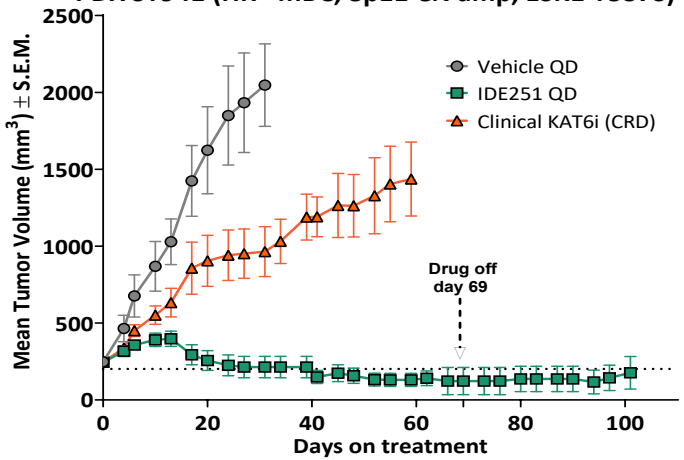
Wide biochemical selectivity window

IDE251 KAT-family in vitro binding



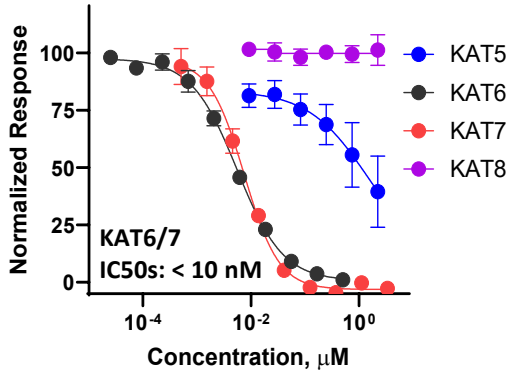
Durable anti-tumor activity

PDX ST941 (HR+ mBC, 8p11 CN amp, ESR1 Y537S)



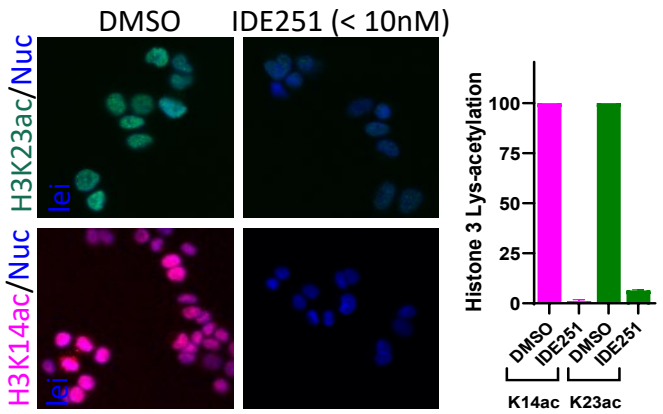
Strong and selective cellular target binding

IDE251 KAT family cellular occupancy assay

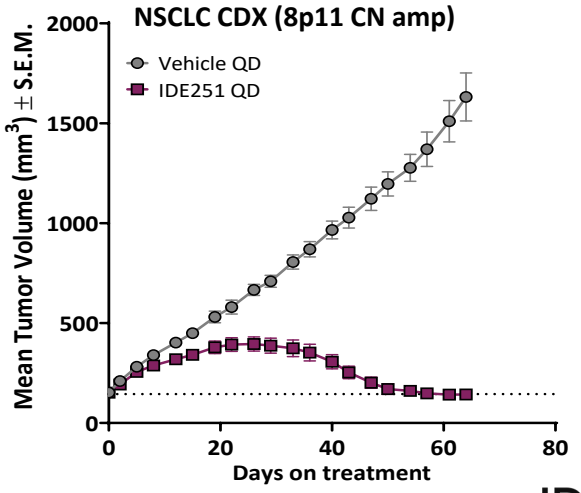


On-target Kac modulation

ER+ mBC model



NSCLC CDX (8p11 CN amp)



(1) IND-enabling studies ongoing with IND-filing targeted in H2 2025
DC = development candidate, CN = Copy Number, mBC = Metastatic Breast Cancer, CDX = Cell Line-Derived Xenograft, QD = Once Daily

Building a Fully-Integrated Biotech in Precision Medicine Oncology

Foundational Potential First-in-Class Clinical Pipeline and Drug Discovery Platform

CLINICAL PROGRAMS	DEVELOPMENT CANDIDATES	PRECLINICAL PROGRAMS
Ph 2/3 – Darovasertib (PKC) [1] Ph 2 – IDE397 (MAT2A) [1] Ph 1 – IDE849 (DLL3 ADC) [2] Ph 1 – IDE275 (Werner Helicase) [3] Ph 1 – IDE161 (PARG) [1] Ph 1 – IDE705 (Pol Theta Helicase) [3]	IDE892 (PRMT5) – Targeting IND Mid-Year 2025 IDE034 (B7H3/PTK7 Bi-Specific ADC [4]) – Targeting IND H2 2025 IDE251 (KAT6/7) – Targeting IND H2 2025	Multiple Potential First-in-Class Programs Advancing
6 Clinical Programs	Targeting 3 IND Filings	

Darovasertib Registration-Enabling Trial with Potential Accelerated Approval in HLA-A2(-) MUM and Ph3 registrational trial targeted in Neoadjuvant UM is tractable for commercial execution and provides path to potential product revenue to reinvest in broad *first-in-class* pipeline

Potential First-in-Class Precision Medicine Oncology Pipeline, including Darovasertib (Ph2/3), IDE397 (Ph 2), IDE849 (Ph1), IDE275 / GSK959 (Ph 1), IDE161 (Ph 1), IDE705 / GSK101 (Ph 1), IDE892 (IND-enabling), IDE034 (IND-enabling), and IDE251 (IND-enabling)

Strong Balance Sheet with ~$1.1B[5] and opportunity for milestone payments with cash runway into at least 2028

Pharma Collaborations including Pfizer, Gilead, Merck, Hengrui, and GSK partnership with ~$2 billion[3] in potential milestones

(1) Clinical Trial Collaboration and Supply Agreements, independently with Pfizer (Darovasertib + Crizotinib), Gilead (IDE397 + Trodelvy®), and Merck (IDE161 + KEYTRUDA®); IDEAYA retains all commercial rights to its products
(2) IDE849 (SHR-4849): DLL3 Top1i Antibody Drug Conjugate. Exclusive license agreement with Jiangsu Hengrui Pharmaceuticals Co., Ltd for worldwide rights outside of Greater China
(3) IDE705 (GSK101) Pol Theta Program Cost Share = 100% GSK with ~$1B Milestones and WW Royalties; IDE275 (GSK959) Werner Helicase Program Cost Share = 80% GSK / 20% IDEAYA with ~$1B Milestones, 50/50 US Profit Share and Ex-US Royalties
(4) IDE034: B7H3/PTK7 Top1i Bispecific ADC development candidate. Exclusive worldwide licensing and option agreement with Biocytogen
(5) Includes aggregate of $1.1 billion of cash, cash equivalents and marketable securities as of December 31, 2024 (Unaudited)